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THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

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BENGT KNUTSSON, "BISSHOP OF ARUSIENS", AND HIS "LITIL BOKE FOR THE PESTILENCE".[1]

By K. F. RUSSELL,
Melbourne.

THE various visitations of the plague to Europe and England were productive of a very extensive output of literature on its cure and prevention. A large proportion of the early medical manuscripts and printed books were devoted to the study of the plague. During the fifteenth and sixteenth centuries probably more books on this subject were published than on any other. Few of these were original, the rest consisting of reissues, in whole or in part, of the more popular treatises, translated into many languages, new editions usually coinciding with a fresh outbreak of the plague.

Of special interest are those which appeared in English. One of the earliest books to be published in English in England will form part of this memoir.

This book, of which the only complete copy in existence is in the John Rylands Library, Manchester, is a small volume of nine leaves printed in London about 1485 by William de Machlinia.

The author, Bengt Knutsson, was elected Bishop of Västerås near Stockholm in Sweden in 1461. He had apparently received a medical training because he says he practised medicine at Montpellier in France. He probably wrote his treatise about 1461-1463, since Sweden had been devastated by the plague several times in the decade preceding this date—in 1450-1455 and again later in 1464 and 1465. The book was published in Latin about the same time as it appeared in English, and went through many editions, the last being in 1722, when it appeared in an edition of Thomas Phaer's writings.

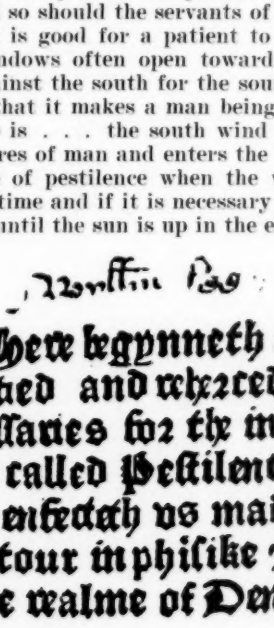
The book itself consists of a brief discussion of the causes of the plague as far as the author's knowledge went, rules of hygiene of a very shrewd nature and some remedies for the prevention of the disease, the latter being very empirical.

He was aware of the contagious nature of the plague as will be seen in the following extracts, these extracts being rendered into a more modern phraseology.

[1] Accepted for publication on May 16, 1939.

"Pestilence sores are contagious because of infected humours in bodys and the reek or smoke of such sores is venemous and corrupts the air. And therefore one should flee from such persons who are infected. In pestilence time nobody should stand in a great press of people because some man present may be infected. Therefore wise physicians in visiting sick folk should stand away from the patient holding their face towards the door or window, and so should the servants of sick folk stand.

"Also it is good for a patient to change his chamber every day and to have the windows often open towards the north and east and to shut the windows against the south for the south wind has two causes of putrifaction. The first is that it makes a man being whole or sick feeble in his body. The second cause is . . . the south wind grieves and hurts the heart because it opens the pores of man and enters the heart. Therefore it is good for a whole man in time of pestilence when the wind is in the south to be within the house in daytime and if it is necessary for a man to go out then let him abide in his house until the sun is up in the east passing southward."


Here begynneth a litil boke the whiche traytied and reherced many gode thinges necessaries for the infirmite a grete sekenesse called Pestilence the whiche oftentimes infectath vs made by the most expert Doctour in philike Bisshop of Aruliens in the realme of Denmark Ac

In his advice on hygiene he mentions several measures which would undoubtedly assist in preventing infection:

"Now it should be written by what means a man may preserve himself from the pestilence. First see the writings of Jeremy the prophet that a man ought to forsake evil things and do good deeds and meekly to confess his sins for it is the highest remedy in time of pestilence, penance and confession is to be preferred to all other medicines. It is a good remedy to leave the infected place but some may not profitably change their places, therefore as much as possible they should eschew every cause of putrifaction and stinking, especially every fleshly lust with women is to be eschewed. Also the southern wind which is naturally infective. Every foul stink is to be eschewed, stables, stinking fields, ways and streets, stinking dead carrion, and especially stinking water which in many places is kept two days or two nights.

"From this cause some die in a house where such things are kept, and in another house none die. Therefore keep your house so that an infected air will not enter, for an infected air will cause putrifaction in places and houses where folk sleep. Therefore let your house be clean and make a clear

fire of flaming wood. Clean your house with fumigation of herbs, that is, with leaves of the Bay tree . . . it is wholesome that you wash your mouth, face, eyes and hands often during the day with rose water meddled with vinegar, and if you have no rose water use vinegar."

He has many instructions for the letting of blood as a precautionary measure against infection and also for the letting therapeutically.

ORDERS,

THOVGHT MEET

BY HIS MAIESTIE, AND His Priuy Councell, to bee executed throughout the Counties of this Realm, in ſuch Townes, Villages, and other places, as are, or may bee heerafter, infected with the Plague, for the ſtay of further increaſe of the ſame.

and for doubt 1630

ALSO,

An Aduice ſet downe by the beſt learned in Phyſick within this Realme, containing ſundry good Rules, and eaſie Medicines, without charge to the meaner ſort of people, as well for the preſeruation of his good Subjects from the Plague before Infection, as for the curing and ordering of them after they ſhall be infected.

LONDON,
Printed by BONHAM NORTON and IOHN BILL, Printers to the King's moſt Excellent Majeſtie. 1625.

"Letting of blood once a month may be done except when age or other things forbid it, such as pilgrims or feeble persons which have the flux. . . . Before eating and after incision of the Basilica be merry and drinke wine or ale or beer temperately."

This book, as we have seen, served as the original for many successive treatises, and very similar instructions, but with a somewhat more complete discussion on the physical signs and symptoms, appeared in the first medical book to be printed in Scotland in the local vernacular.

This book by Dr. Gilbert Skeyne was "Imprentit at Edinburgh be Robert Lekprevik anno do. 1568" and its quaint title was: "Ane Breve Descriptioun of the Pest quhairin the Causis, signis and sum speciall preseruatioun and cure thairof ar contenit."

The official legislation for persons afflicted with the plague and with the ordering of places and towns so affected would appear to have been somewhat late in appearing in England. One of the earliest of these official rules appeared as "Orders thought meet by Her Majestie etc." printed in London 1588-1590. This was reprinted several times and the title page of the 1625 edition forms an illustration to this paper.

Some of the orders in this book are reprinted below:

"Item they shall cause to bee appointed in every parish, as well infected as not infected, certain persons to viewe the bodies of all such as shall die, before they bee suffered to bee buried, and to certifie to the minister of the church and church-warden or other principall officers, of what probable disease the said persons dyed . . . and in case the said viewers either through favour or corruption, shall give wrong certificate, or shall refuse to serve being thereunto appointed then to cause them to bee punished by imprisonment, in such sort as may serve for a terror to others.

"Item the houses of such persons, out of the which there shall die any of the plague, being so certified by the viewers . . . to bee closed up on all parts during the time of restraint, viz. six weeks after the sicknesse be ceased in the same house in case the said houses so infected shall be within any towne having houses neere adjoyning to the same. And if the infection happen in houses dispersed in villages and separated from other houses, and that of necessitie for the serving of their cattell the said persons cannot continue in their houses, then they be nevertheless restrained from resorting into companie of others, eyther publickely or privately, during the said time of restraint, and to weare some marke in their uppermost garments, or beare white rods in their hands at such time as they shall goe abroad. And furthermore some speciall marke shal be made and fixed to the doores of every of the infected houses and where any such houses shal be Innes or Alehouses, the signes shal be taken downe for the time of the restraint, and some crosse or other mark set upon the place thereof to be a token of the sicknesse."

That statistics should be kept is ordered by the following:

"Item the Ministers and Curates, and the Churchwardens in every parish shall in writing certifie weekely to some of the Justices residing within the Hundred or other limit where they serve, the number of such persons as are infected and doe not die, and also of all such as shall die within their Parishes, and their diseases probable whereof they died, and the same to be certified to the rest of the Justices at their assemblies, which during some

convenient time would bee every one and twenty dayes, and thereof a particular booke kept by the Clerke of the Peace or some such like.

"Item for that the contagion of the plague groweth and increaseth no way more, then by the use and handling of such clothes, bedding and other stuffe as hath been worne and occupied by the infected of this disease; the said Justices shall in the places infected take such order, that all the said clothes and other stuffe, so soone as the parties diseased of the plague are all of them either well recovered or dead, be either burnt and cleane consumed with fire, or else ayred in such sort as is prescribed. And for that peradventure the losse of such apparell, bedding and other stuffe to be burnt, may bee greater then the poore estate of the owners of the same may well beare . . . then the sayd Justices out of such collections as are to bee made within their counties for the reliefe of the poorer sort that bee infected, allow also to them such sum or summes as to them shall be thought reasonable, in recompense of the losse of their sayd stuffe."

During the Great Plague of London in 1665 the Lord Mayor published additional orders to those mentioned above. These were an amplification of the original orders by the King and Privy Council, and they aimed at a more rigid sequestration of the sick in order to prevent the spread of the infection.

There were rules made for the burying of bodies. "No corpse dying of infection shall be buried, or remain in any church in time of common prayer, sermon, or lecture. And that no children be suffered at time of burial of any corpse, in any church, churchyard, or burying place, to come near the corpse, coffin or grave. And that all the graves shall be at least six feet deep."

Hackney coaches which had been used to convey infected persons to the pest house had to be well aired and let stand for six days before being used again.

There were rigid rules against the meeting together of the people: "That all plays, bear-baitings, games, singing of ballads, bucklerplay, or such like causes of assemblies of people, be utterly prohibited, and the parties offending severely punished by every Alderman in his ward.

"That all public feasting, and particularly by the Companies of the City, and dinners at taverns, ale-houses, and other places of common entertainment, be forborne till further order and allowance; and that the money thereby spared, be preserved and employed for the benefit and relief of the poor visited with the infection.

"That disorderly tipling in taverns, ale-houses, coffee-houses and cellars be severely looked unto, as the common sin of this time, and greatest occasion of dispersing the plague. And that no company or person be suffered to remain or come into any tavern, ale-house, or coffee-house, to drink after nine of the clock in the evening."

The illustration of the first page of "A Litil Boke for the Pestilence" is taken from the facsimile issued by the John Rylands Library, Manchester.

BIBLIOGRAPHY.

Bengt Knutsson: "A Litil Boke for the Pestilence", London Ca. 1485. John Rylands Facsimile Number 3, Manchester, 1910.

"Orders thought meet by His Majestie and Privy Council to bee executed . . . for the Plague", London, 1625.

Daniel Defoe: "The History of the Great Plague in London in the Year 1665", London, 1819.

J. D. Comrie: "The History of Scottish Medicine", two volumes, second edition, 1932.

ARTIFICIAL COLLATERAL CIRCULATION TO THE HEART: SOME CRITICAL COMMENTS ON ITS VALUE.[1]

By E. S. J. King,
Surgeon to Out-Patients, Royal Melbourne Hospital, Melbourne.

During the last two decades the importance and frequent occurrence of disturbances of the vascular supply to the heart have become thoroughly appreciated. The myocardial dysfunction which results from vascular disorders has commanded the attention of physiologists, pathologists and clinicians; and the interest invoked is indicated by the yearly increasing increments to the literature. In view of the very considerable progress that has been made in surgery during recent times, not only with regard to general problems, but also with regard to the management of diseases of the heart and pericardium, it is natural that attempts should have been made to deal with this problem of myocardial disturbance resulting from coronary disease.

A number of different methods has been suggested to deal with this condition; but one of the most important, if not for its intrinsic worth, because of the publicity it has received, has been the attempt to produce a new circulation for the organ by way of adhesions induced between the heart and other structures, such as the omentum, pectoral or other muscles, mediastinal tissues, the pericardium or the lung. The observations that suggested the use of such a method and the gradual development of the operative technique have been thoroughly discussed by Beck, O'Shaughnessy and others, and need not be entered into in detail here.

The use of such methods has given rise to a great deal of discussion, and though there is as yet probably insufficient evidence on which to base a satisfactory conclusion, there are two very definite but opposed groups of opinion. On the one hand, investigators, who have been particularly instrumental in devising technical procedures *et cetera,* consider that the evidence derived from the experimental work on animals and also from clinical cases in which this procedure has been used, strongly demonstrates its value. On the other hand, other observers are doubtful whether the amount of blood that can be supplied to the heart through such new-formed vessels, can possibly be of value in improving the blood supply. A number of observations which appear to have a very important bearing on this question are given in this paper.

Historical.

Changes in the coronary vessels have been recognized for a very considerable time, and were noted in the eighteenth century by Jenner, Parry, Baillie and Thebesius amongst others (Hodgson). Though their observations seem to have been almost forgotten for nearly a century, it is noteworthy

[1] Accepted for publication on February 14, 1940.

that Jenner appreciated that there was a relationship between diseases of the vessels, particularly calcification, and interference with cardiac function.

It was not, however, until the later part of the last century that the relationship was definitely enunciated and became generally appreciated. It was noted in 1876 and 1878 by Stevenson and Ménard, and later was more completely described by Weigert, Ziegler and others. This relationship was at first only a pathologically recognized one, but in the last thirty years the clinical features of myocardial damage following vascular occlusion have

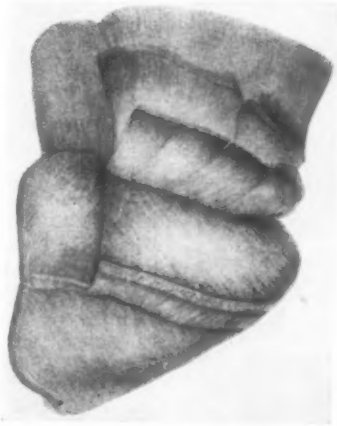


FIGURE I. Reproduction of a drawing from Jules Cloquet's "*Anatomie de l'homme*", Paris, 1828, showing the layers of muscles in the heart wall. This is of historical interest, and although not indicating the distribution of the layers, it shows that the structure of the heart wall was appreciated more than a century ago.

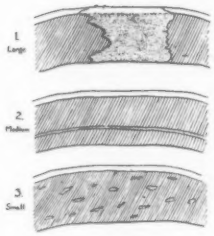


FIGURE II. Diagrammatic representations of portion of the ventricular wall showing different types of lesions. 1. An irregular infarct involving a localized area, but the whole thickness of the wall; this is usually due to obstruction of a large coronary vessel. 2. A laminar lesion, shown here as a contracted scar, which is usually due to interference with the blood flow through a branch or branches of the main coronary vessels. 3. Discrete small areas of fibrosis resulting from obstruction to, or ineffective circulation in, very small vessels.

become more and more widely understood, until now it is a frequently diagnosed condition.

For a considerable time it has been thought that the distribution of the changes in cardiac muscle occurs in two forms—either a diffuse and punctate one, or a localized wedge of tissue comparable with an infarct of other organs—and that these were the important and indeed sole lesions.

In 1874 Fagge described, before the Pathological Society of London, a number of cases in which cardiac scars were of a laminar form. Although similar observations have occasionally been either described or figured in illustrations, this phenomenon appears since that time to have attracted little or no attention. Recent investigations by Robb, Lowe and others have suggested that the various layers of muscle in the ventricular wall resemble the individual muscles of the limbs in that they have a separate blood supply. Certain branches of the coronary arteries can be shown to supply practically exclusively such layers or parts of them.

Although the writer is satisfied that the evidence for this opinion is good, at the same time it is not essential for the present argument. The important feature is the peculiar distribution of the affected muscle and, incidentally, the scar tissue.

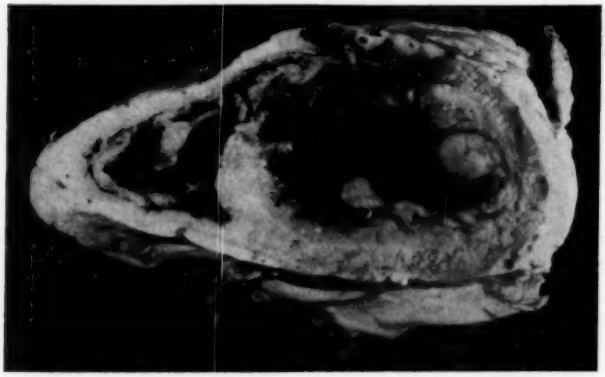


FIGURE III. Photograph of a cross-section through the ventricles showing an infarct in the anterior wall of the left ventricle (viewed from below). The anterior descending branch of the left coronary artery containing a thrombus is visible and there is a beginning rupture through the wall in this region.

MORPHOLOGICAL OBSERVATIONS.

Although in post-mortem specimens obtained from recent cases of coronary disease areas of infarcted tissue may be seen, in the majority of cases the principal evidence of previous ischæmia is in the form of scar tissue. This is found in three different distributions (Fig. II).

1. In some cases, particularly in recent occlusions of relatively large vessels, an area involving the whole thickness of the wall, but more or less localized to one zone, may be affected. Such areas resemble infarction of other organs. It is in such cases that rupture usually occurs, or perhaps it should be said that in cases of rupture this type of myocardial involvement is found (Figure III). That such an infarcted area is due to the obstruction of a large vessel is shown partly by the discovery *post mortem* of an obstruction, perhaps a thrombus, in such a vessel, but also by the discovery of such lesions when a large vessel is ligated in a dog's heart.

2. The second type is that in which the scar tissue has a laminar arrangement. Although this is a common finding, it has, as previously mentioned, escaped

FIGURE IV. Photograph of a heart showing an aneurysm of the left ventricle. The laminar scar near the endocardium, with relatively normal-looking muscle in the peripheral part of the wall, is shown.

notice. Careful examination of these scars with reconstruction from serial sections has shown that they correspond in distribution very closely to one or other of the various individual heart muscle layers. These laminar scars may be found in a considerable proportion of the hearts either of individuals who have had symptoms of coronary disease or of individuals, without such symptoms, whose hearts show definite evidence of occlusive change in the vessels.

Although these scars, or in recent cases areas of infarction, may be found when the heart chambers are opened in the ordinary way by longitudinal incisions (Figures V and VI), it has been found that it is very easy to overlook them. When, however, the organ is sectioned at right angles to its long axis, the incisions being made from one-half to one inch apart, these laminar scars are readily demonstrable. Several of these are illustrated in Figures VII, VIII and IX.

FIGURE V. Photograph of a heart, showing a recent infarct of the laminar type.

FIGURE VI. Photograph of a heart, showing a healed laminar subendocardial scar.

It has been shown recently by Lowe that if one of the smaller branches of a coronary artery is separately injected (for example, with a differently coloured material from that used for the remaining vessels of the heart), a muscle layer or portion of it will be found to be differentially injected. It would seem, therefore, that a diminution in the passage of blood through one or more of the smaller vessels is responsible for a laminar distribution of infarction. It must be appreciated here that it is very necessary to take into consideration the physiological pathology of the circulation as well as the morphological changes in the

vessels. Thus it does not necessarily mean that these smaller vessels, which, with experience, can be readily recognized, are themselves occluded. Obstruction to a large vessel will cause lowering of the blood pressure beyond this obstruction. If portion of the corresponding arterial tree receives some augmentation of its blood content from a collateral supply, then only a small part of the area supplied by the main vessel will become ischæmic. In this way obstruction to a large vessel may give a result similar to that produced by obstruction to a smaller artery.

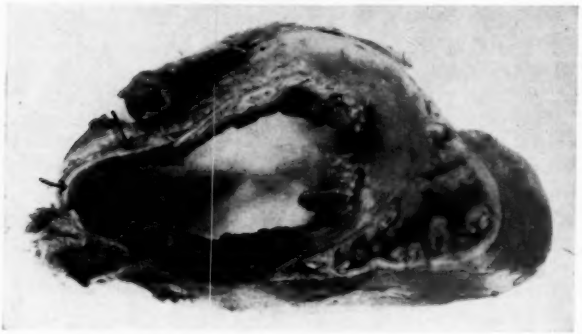


FIGURE VII. Photograph of a cross-section through the ventricles, showing a laminar scar in the wall of the left ventricle.

Obviously the result depends on the rate of development of the obstruction, and this is discussed later. However, whatever the mode of formation of these scars, their general distribution and nature are definite, and it is on these that subsequent argument is based. These laminar scars are to be found very well developed in cases of aneurysm of the heart (Figure IV). Of course, they are often found when there is no dilatation, but if a sufficiently important layer becomes ischæmic, or if more than one layer is affected at a time, it is easily understandable that the cardiac wall would be unable to withstand the endocardiac ventricular pressure and so would distend.

3. In the third type a number of small, more or less discrete fibrous areas may be scattered throughout the

FIGURE VIII. Photograph of a cross-section through the ventricles showing well-defined laminar scars in the wall of the left ventricle. (Courtesy of Dr. T. E. Lowe.)

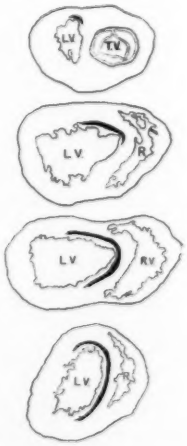


FIGURE IX. Drawings from a series of transverse sections through the ventricles showing the extent of a scar at different levels. L.V. = left ventricle; R.V. = right ventricle; T.V. = tricuspid valve.

myocardium, and these are obviously associated with obstruction to small coronary arteries or arterioles in different parts of the heart wall.

PATHOLOGICAL PHYSIOLOGY.

Although there has been a great deal of discussion for many years regarding the presence of a significant collateral circulation in the heart itself, a considerable amount of information has gradually been collected indicating that such communications do exist between vessels and their branches. It would seem that they are of value in normal circumstances only to a limited extent, and cannot play any important role when a large vessel is suddenly obstructed. When obstruction occurs gradually, however, there is time for dilatation of the anastomotic vessels, and complete obstruction of even a main coronary branch can develop without any apparent interference with the structure or function of that part of the myocardium which was originally supplied by it. There are anastomoses also between branches of the coronary vessels and extracardiac branches, particularly in the region of the great vessels. That these may provide satisfactory circulation to the heart is shown by those cases in which, at post-mortem examination, complete occlusion of all the large coronary vessels has been demonstrated. This was observed and remarked upon by Thorel in 1903, and has been described by a number of subsequent observers. It is not an excessively rare phenomenon, as the writer has observed four such cases. As stated, it was such observations that suggested the artificial production of a collateral circulation.

The passage of blood through vessels and the direction of its flow depend on definite physical laws: the mere presence of blood-containing vessels in a region does not necessarily presuppose an adequate blood supply to that area. Three factors are necessary: (i) an adequate head of pressure in the vessels delivering blood to the region, (ii) sufficient calibre of the anastomosing vessels, and (iii) adequate stimulus to the development of the new blood supply, that is, to the passage of blood in the required direction.

In the heart itself, blood for an ischæmic area will be obtained from adjacent coronary vessels, and unless the main branches are blocked so that the pressure in these vessels is lowered, there will be sufficient pressure to deliver blood to the affected region.

Some of the anastomosing vessels are of sufficient size to carry a considerable quantity of blood, but in the more peripheral parts of the heart communicating vessels are small. In addition to this, since an atheromatous degeneration usually affects many vessels, it must be realized that, if blockage in the circulation in one area is due to such an atheromatous condition, neighbouring arteries may also be involved and thus the available supply may be insufficient. In addition the actual anastomotic vessels themselves may be the site of the atheromatous process.

Even when the existing vessels are inadequate for the supply of a region, provided that the ischæmia develops slowly, they may become dilated and ultimately become of sufficient diameter. Disturbance of the normal flow to an area results in the accumulation of products of tissue activity and of tissue injury in these regions, and these will give rise to dilatation of vessels in the neighbourhood and, amongst others, the collateral branches. This constitutes the "demand" for an increased blood supply. When this blood

supply is maintained by these vessels for some time, they become permanently enlarged and develop the morphological characters of larger vessels.

This question of the gradual development of collateral vessels is of great importance when the effects of vascular obstruction are considered. Thus a sudden obstruction to a small artery may produce a much more serious effect than a gradually developing block in a larger vessel, since in the second case there may have been time for a collateral blood supply to have formed. This has an important bearing on the statement made previously with regard to the nature of lesions in the heart in relationship to the size of vessels affected; but this need not be elaborated here.

When an artificial collateral circulation is produced in any way, the factors just mentioned determining an adequate supply of blood must again be present.

1. There must be an adequate head of pressure in the vessels which are to supply the new circulation, though, of course, it is immaterial whether these vessels occur in the omentum, pectoral muscle, mediastinal tissue, lung or even in the pericardium itself. It might be thought that, since the pericardium is relatively avascular, it would be unsuitable for such a purpose, but observation of examples of inflammation of the pericardium shows that the small vessels which are present may rapidly become quite large. It is essential that the pressure in these vessels should be greater than it is in the vessels in the affected area of the myocardium because, if there is not a definite "pressure gradient" from the supplying vessels to the supplied area, blood of course will not flow in this direction.

2. The anastomosing vessels in most cases are, at least in the beginning, of the calibre of capillaries, and it is necessary that these should enlarge in order for them to be able to carry a sufficient quantity of blood. This, of necessity, requires some time for a complete development. In addition, it is important that the larger vessels should be morphologically normal, or at least relatively normal.

3. There must be the definite stimulus to the passage of blood towards the myocardium because otherwise the vessels remain small and adhesions may disappear. It is axiomatic that the formation of a circulation can have no possible effect on muscle that has been damaged already, but can influence only muscle which, though receiving a grossly diminished blood supply, has not yet died; or it may prevent the further ischæmic degeneration of muscle fibres.

When a collateral circulation is being established, it is important not only that there should be a "demand" for an increased blood supply on one side of the capillary network, but that this stimulus should be continued. Should the stimulus be inadequate or disappear, then the small vessels will not enlarge, and, indeed, when they are present in adhesions, they, together with the adhesions, may disappear. This disappearance or resolution of adhesions is a common phenomenon, particularly in the peritoneal cavity, but may be observed also in the pleural and pericardial cavities. Experimentally it may be shown that if some structure such as omentum is attached to the normal heart wall, any adhesions that occurred at an early stage may disappear after some time and the structure loses its original attachment to the heart.

DISCUSSION.

For the formation of a collateral circulation to the heart, after the application of some vascular structure to the surface of the organ, the various requirements mentioned above must be fulfilled. From this point of view

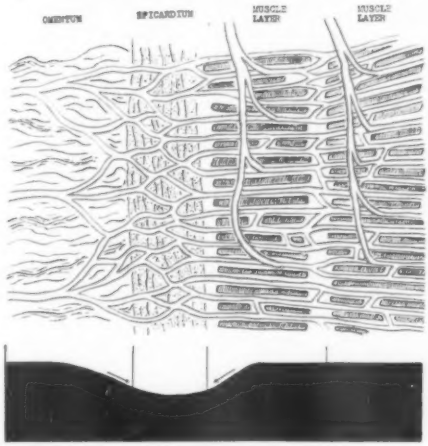


FIGURE X. Drawing of the superficial portion of the ventricular wall, the epicardium and an omental graft to indicate the vascular relationships. When an anastomotic connexion has been made between the two groups of vessels in the epicardial layer, blood will flow both from the myocardial and from the omental vessels to the epicardial layer; from this region it passes into veins. The pressure in the myocardial vessels will be of the same order as that in the omental vascular system. This is shown diagrammatically in the lower portion.

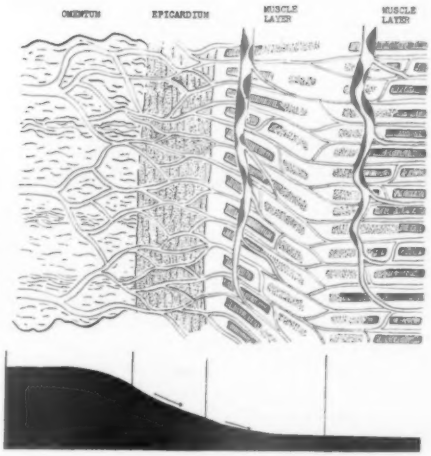


FIGURE XI. Drawing similar to that shown in Figure X. In this case the arteries supplying both the superficial layers are diseased and the blood supply is diminished, with some degeneration of muscle fibres. In this case the pressure in the myocardial region is lower than in the omental vessels (presuming that these are normal), and therefore blood could pass into the myocardium. This is indicated in the subjacent graph.

there are two types of lesion that may be encountered: the type in which the ischæmic tissue is on the surface and that in which it is deeply situated and separated from the surface by an area of relatively normal myocardium.

If the affected tissue is superficial (Figure XI), the blood supply of this portion of the myocardium is poor and the effective intravascular pressure is lower than it is in the vessels of the tissue applied to the surface of the heart.

When the two sets of vessels become united, there will therefore be a definite "pressure gradient" from the extracardiac vessels to those in the myocardium.

If, on the other hand, the surface tissue is in good condition, as in the normal heart, then the pressure in the corresponding groups of vessels is approximately equal (Figure X); thus the blood pressure of both sides of the capillaries will gradually fall toward this region, whence the blood will pass away in the venules and veins. Thus there will not be a passage of blood from one part to the other and, as stated previously, in the absence of some definite stimulus, the adhesions will disappear.

If the lesion is a deep one, then the circumstances, from the point of view of the superficial tissues, are similar to those observed in the normal heart: the pressure in the superficial layer will be equivalent to that in the supplying tissue (Figure XII). The pressure and volume of blood in the

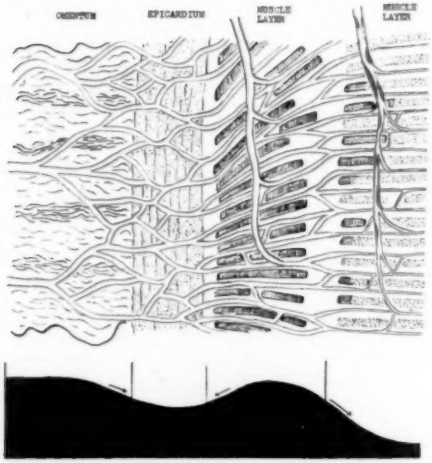


FIGURE XII. Drawing similar to those in Figures X and XI. In this case it is the deeper layer which is deprived of its blood supply, and the damage to muscle suggests that the collateral supply from the superficial layer has been inadequate. The graph indicates that since the pressure in the vessel supplying this layer is of the same order as in the omental vessels, the artificial collateral supply cannot influence the condition of the deeper damaged myocardial layer.

superficial layer may at times be sufficient to give a supply to the deeper ischæmic myocardium, but when this is not so, it is improbable that the presence of a vascular tissue on the surface of the heart will influence the condition of the deeper tissues (Figure XII).

Now if we consider the frequently observed laminar lesion, particularly in view of the fact that it often lies in the deep layers of the myocardium (Figure IX), it is apparent that the operation for the production of a collateral circulation cannot be effective. It may be of some value when the layer comes to the surface of the heart in part of its extent. Reference to the distribution of muscle layers shows that this happens with even the deeper muscles. It is often found, however, that only the deeper or subendocardial portion of such a muscle is affected; and this suggests that adequate blood supply to the superficial part does not, in ordinary circumstances, presuppose a sufficient supply to the deeper portion.

It is important, when a decision is made on the suitability or otherwise of a case for operation from this point of view, that the distribution of the lesion in relationship to the layers of the heart muscle should be determined.

This is not possible at present, although some of the observations made by Robb suggest that it may yet become practicable.

In deep involvement of the myocardium therefore it is the absence of one of the important requirements, that is, the "demand" for a blood supply from the new tissue (omentum, muscle *et cetera*) from the adjacent heart surface, that is responsible for the failure of development of the collateral circulation. It is noteworthy here that some investigators, notably Beck, have emphasized the importance of injuring the superficial part of the heart to ensure the attachment of the graft to the heart wall. This would suggest that a stimulus from ischæmia is not present.

When we consider the actual results of operative treatment, however, we find that the protagonists of this method can present an apparently strong case in its support. The evidence is derived partly from animal experiments and partly from clinical observations.

The various experiments on animals seem to show that ligation of a coronary vessel, after a graft has been applied to the heart, does not produce the result that occurs in the absence of such a graft. There are several reasons, however, why such observations cannot be accepted without critical examination. First, these observations are made on animals with a healthy myocardium; it is noteworthy that the results of experiments are not so striking if aged instead of young animals are employed. Secondly, the vessels of the animals are usually normal, and thus any results cannot be regarded as comparable with changes observed in human beings with diseased vessels. Thirdly, mechanical interference with a vessel almost invariably means some change, usually occlusion, of the accompanying veins. In atherosclerosis the arteries are affected to the exclusion of the veins. When the differences observed in the limbs between simple ligation of a main artery on the one hand and artery and vein together on the other are taken into account, it is clear that deductions made from ligation experiments cannot be accepted unreservedly.

Injection of specimens obtained after the application of a graft to the heart shows that material may pass from an extracardiac structure through vessels into the wall of the myocardium. Very few observations are necessary to show that this gives no real indication whatever of the effective distribution of blood from such a structure in the intact animal. Many of the conclusions drawn from injection experiments are invalidated by this lack of consideration of physical principles and physiological phenomena.

When we come to the clinical cases, however, it would seem that, despite any theoretical considerations, the results justify the use of the method. The writer's experience would agree with this. The important point, nevertheless, is whether the patient's condition improves because of an increased blood supply or for some other reason. Two of the writer's observations suggest that the second explanation is the true one. (i) In some cases in which operation was performed and in which there was some degree of demonstrable cardiac insufficiency, this was shown to be lessened within a day or so of operation: pain was relieved, dyspnœa was improved and slight œdema of the lower limbs diminished. This improvement thus was apparent several days before any collateral circulation could conceivably have developed. (ii) Satisfactory results were obtained principally in those cases in which the heart was

enlarged, and no improvement whatever was found when the heart was small, that is, when it did not fill the pericardial cavity and distend the pericardial sac.

As shown by Felix and O'Shaughnessy, amongst others, insufficiency of heart action, when the pericardium is "tight", is greatly lessened by incision of the parietal pericardium. Normally the pericardium is inextensible, but there is sufficient room in the sac to allow of adequate diastolic filling of the heart, even when it is under considerable stress. If, however, the heart becomes dilated or hypertrophied, without any softening—and thus stretching—of the parietal pericardium, with consequent enlargement of the pericardial sac, the heart becomes relatively too large for the pericardium. The pericardium therefore becomes "tight" and thus diastolic filling of the heart cannot occur satisfactorily. If now the pericardium is incised, the heart is able to dilate to a greater extent during diastole and thus its action becomes more efficient. The observation mentioned above would be explained on this hypothesis of the mode of action of operation much more satisfactorily than on that of the development of a collateral circulation.

It is possible that the heart and its vascular supply may be influenced by the procedure in other ways, for example, by alterations in the nervous control; but, although a great deal of information is accumulating with regard to these nerves and their physiological action, it is not possible at present to make any statement of value one way or the other. It is noteworthy, and possibly significant, that the clinical cases in which patients derive most benefit from operative interference are those in which pain is a predominating feature. This question is a complex one, raising many knotty points, and must remain outside the scope of this paper.

The application of a graft to the heart may be of value in other ways. Thus it may be shown to be of use in cases of incipient aneurysm, but in such circumstances it would be partly at least a question of a supporting bulk of tissue rather than any question of an improved blood supply.

There are other procedures that have been suggested for the relief of the results of coronary disease. The most important of these is ligation of the coronary sinus, but though this is probably the most effective method of improving the capillary supply to muscle fibres, it does not require more than mention here.

CONCLUSIONS.

1. An examination of hearts, the seat of coronary disease, shows that grossly involved tissue or scars often have a laminar form.

2. These laminar areas or scars probably correspond to muscle planes or parts of them and usually lie in the deeper layers of the heart, that is, are separated from the epicardial surface by a band of relatively normal myocardium. Their form, however, is the important feature, and this importance is independent of their relationship to anatomical layers.

3. The passage of blood from one group of vessels to another depends on a difference in intravascular pressure; thus (*a*) if the heart is normal, such flow will not occur from grafts towards the heart; (*b*) if the superficial layer of the heart is ischæmic, blood may flow from an extracardiac structure to and thus supply the heart muscle; (*c*) if the affected tissue lies deep in the heart wall, blood will not flow from an extracardiac structure to the superficial layer and thus cannot reach the affected tissue.

4. In many cases, therefore, an artificial collateral circulation to the heart cannot be effective.

5. Experimental work, supporting the value of a surgically produced collateral circulation, must be critically examined before its significance can be assessed.

6. Clinical cases in which there is an indubitable improvement after operation have been observed, but such improvement is almost certainly due to factors other than the formation of a new circulation.

BIBLIOGRAPHY.

C. S. Beck and V. L. Tichy: "The Production of a Collateral Circulation to the Heart. 1. An Experimental Study", *American Heart Journal*, October, 1935, Volume x, page 849.

C. Hilton Fagge: "A Series of Cases of Fibroid Disease of the Heart", *Transactions of the Pathological Society of London*, 1874, Volume xxv, page 64.

W. Felix: "*Tätigkeit des Herzens bei verscheiden starkem Druck auf seine Oberfläche*", *Deutsche Zeitschrift für Chirurgie*, 1930, Volume cxxxix, page 1.

J. Hodgson: "A Treatise on the Diseases of the Arteries and Veins", 1815.

"Edward Jenner: His Life, His Work and His Writings", *The British Medical Journal*, May 23, 1896, page 1245.

T. E. Lowe: "The Significance of Myocardial Scars in the Human Heart", *The Journal of Pathology and Bacteriology*, Volume xlix, July, 1939, page 195.

V. Ménard: "*Cas de rupture spontanée du coeur*", *Bulletins de la société anatomique de Paris*, 1878, Volume liii, page 61.

L. O'Shaughnessy and L. McCunn: "Surgical Treatment of the Hypertrophied and Dilated Heart", *Veterinary Journal*, Volume cxii, January, 1936, page 6.

L. O'Shaughnessy, D. Slome and D. T. Davies: "Surgical Revascularisation of the Heart", *The Lancet*, March 18, 1939, page 617.

C. H. Parry: "An Enquiry into the Symptoms and Causes of Syncope Anginosa, Commonly Called Angina Pectoris", 1799.

J. S. Robb: "The Structure of the Mammalian Ventricle", *Medical Women's Journal*, Volume xli, March, 1934, page 203.

J. Stevenson: "Endoarteritis Chronica Deformans, and its Relation to Rupture of the Heart", *Edinburgh Medical Journal*, Volume xxii, September, 1876, page 216.

A. C. Thebesius: "*Dissertatio medica de circulo sanguinis in corde*", 1716.

C. H. Thorel: "*Pathologie der Kreislauforgane*", *Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie des Menschen und der Tiere*, Volume ix, 1903, page 559.

C. Weigert: "*Über die pathologischen Gerinnungsvorgange*", *Virchows Archiv für pathologischen Anatomie und Physiologie und für klinische Medizin*, Volume lxxix, 1880, page 87.

E. Ziegler: "*Lehrbuch der allgemeinen und speciellen pathologischen Anatomie und Pathogenese*", 1887.

A METHOD FOR THE CONTINUOUS RECORDING OF CEREBRO-SPINAL FLUID PRESSURE LEVELS: A PRELIMINARY REPORT ON THE EFFECTS OF INTRAVENOUS THERAPY IN THE TREATMENT OF CEREBRAL ŒDEMA.[1]

By F. F. Ellis,

Flight Lieutenant, Royal Australian Air Force; George Adlington Syme Research Scholar, 1937.

The results of the treatment of cerebral trauma by modern methods have conclusively proved the value of the prevention of cerebral œdema during the patient's stay in hospital. This has been borne out by a mortality rate of 10% or less since the introduction of these methods to Lewisham Hospital, Sydney, where I first conducted these studies.

The relief of cerebral œdema may be approached by one of two ways: either by lumbar puncture or by intravenous injections of hypertonic fluids. Lumbar puncture with the use of a simple tubing manometer is a very simple procedure which enables the intracranial tension to be measured quantitatively and the pressure to be lowered to any desired positive level. According to circumstances, one or several such lowerings of pressure may be necessary to prevent the development of cerebral œdema.

When intravenous therapy, however, is used, unless a lumbar puncture is done previously and concurrently there is no knowledge either of the existing intracranial tension or of the influence of the particular solution on it. Thus the method is empirical and there is no standard by which to judge the effects of intravenous hypertonic injections other than by an observation of the clinical effects. It is desirable, therefore, that an investigation should be made to determine quantitatively the influence of various hypertonic solutions on patients of different sexes and ages suffering from various degrees of acute and subacute cerebral trauma.

My interest led me into an investigation of the quantitative effects of intravenous therapy in the treatment of head injuries and of post-operative cerebral œdema. Hence there developed this instrument which makes an actual graphic record of cerebro-spinal pressures over short periods of time. It has been called the "cottunometer", from the old term *liquor cotunnii* applied to cerebro-spinal fluid. Its construction is as follows (Figures Ia and Ib).

There are two compartments, as shown, the smaller one being again divided into two sections. A section, marked A, houses the camera film; this film is driven by a small electric motor, which occupies the section B.

[1] Accepted for publication on September 13, 1938.

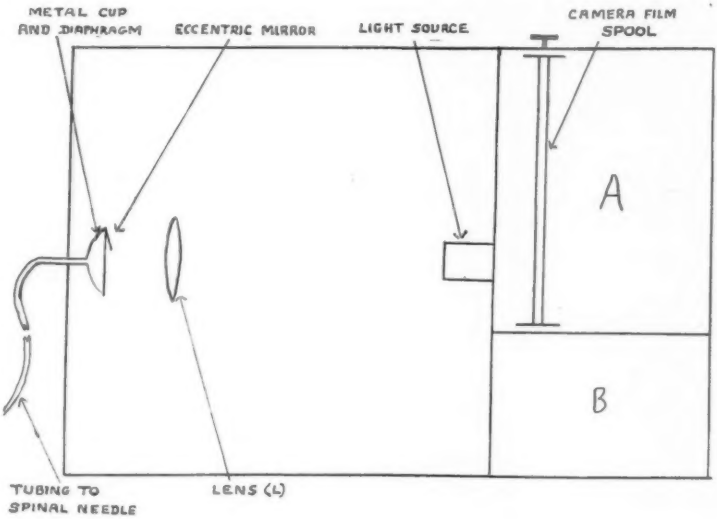


FIGURE IA. Structure of the "cottunometer" viewed from above.

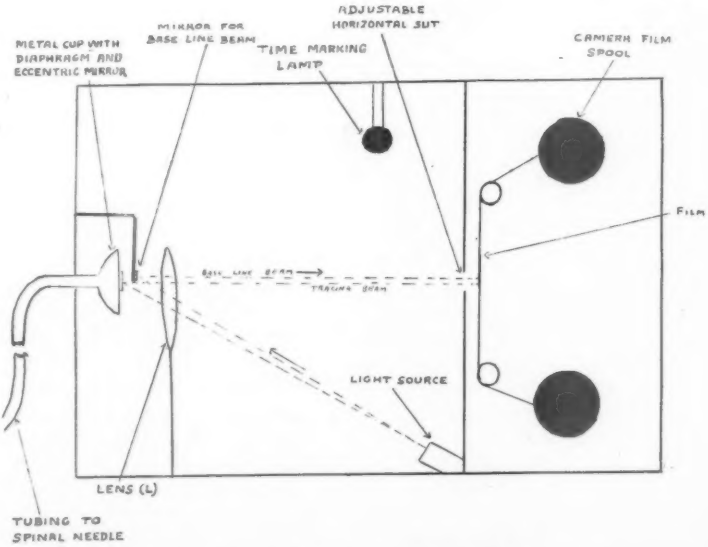


FIGURE IB. Structure of the "cottunometer" viewed from the side.

In the larger compartment is the light source which throws a powerful fine vertical beam of light from the camera end of the box to the further end, where a fine metal tubing pierces the box to end in a small metal cup over which is stretched a thin rubber membrane. On this membrane is placed eccentrically a tiny mirror. The light source is so adjusted that the fine vertical beam of light passing through a convex lens (L) impinges on the tiny mirror and is reflected therefrom. The reflected light passes back to the camera end of the box, and passes through a fine adjustable horizontal slit to reach the moving sensitive film as a fine point of light. Now, any alteration in pressure behind the diaphragm will be transmitted to the tiny mirror on it and hence, magnified by the beam of light, to the camera film.

As the direction of any variation in the beam is horizontal and the movements of the camera film are vertical, the result, on development of the film, is a finely traced graph, examples of which will be shown below. In addition, a tiny fixed mirror is suspended in the beam from the light source immediately above the mirror that is attached to the diaphragm, and is so adjusted that it gives a base-line to the developed film.

Furthermore, placed above the camera end of the large compartment is a small electric lamp which flicks on momentarily each minute by virtue of contact made at each minute revolution of the camera motor. This results in a thin black streak across the developed film at each minute interval, thus giving the time relationship to the finished graph.

Now, the metal tubing from the diaphragm cup passes to the exterior and can be connected to the tubing from a lumbar puncture needle by means of a record fitting adaptor. In this way records can be taken of the cerebro-spinal pressure and of consequent variations in it as transmitted via the tubing from the patient's spinal sac to the diaphragm.

Before this instrument was used clinically it had to be calibrated, and this was done by taking an actual record whilst the pressure behind the diaphragm was raised in increments of twenty millimetres of cerebro-spinal fluid pressure with a water manometer. Two graphs of this are reproduced, and it will be noted that the relationship between deflection and increasing tension is practically linear (Figures IIA and IIB). This is advantageous, as any pressure on an ensuing record can be estimated by comparison with the calibration record. The relationship for the records reproduced is 20 millimetres deflection to 100 millimetres of cerebro-spinal fluid pressure.

The technique of the use of the "cottunometer" is briefly as follows.

The patient being placed in the recumbent posture as for an ordinary spinal puncture, the "cottunometer" is placed at the bedside at a sufficiently high level for the metal tubing to be between twenty and thirty centimetres above the lumbar region.

The spinal puncture is performed and the pressure is taken by an ordinary open tubing manometer. This pressure is noted. Then the distal end of the tubing of the diaphragm cup is connected to the lumbar puncture needle and the camera motor is set in motion. A shutter over the slit in front of the film and a switch on the camera motor make it easy to hold the recording in abeyance at any stage should this be necessary. The shutter is also useful as a signal, for if it is rapidly closed and opened, it breaks the record at the required moment on the developed film.

Each film takes about eighteen minutes to run through, during which time any investigations can be made or injections can be given, the effects of which will be impressed on the film.

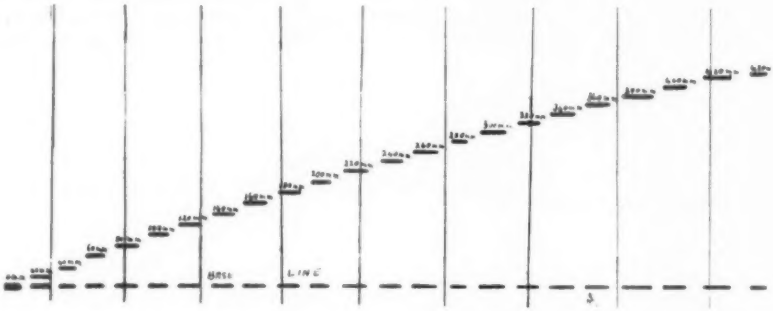


FIGURE IIA. Calibration tracing showing the base-line, the minute time markings and the effects of increments of pressure on the developed film. A rise of 100 millimetres of water in pressure causes a rise of 20 millimetres on the tracing. The end result is almost linear.

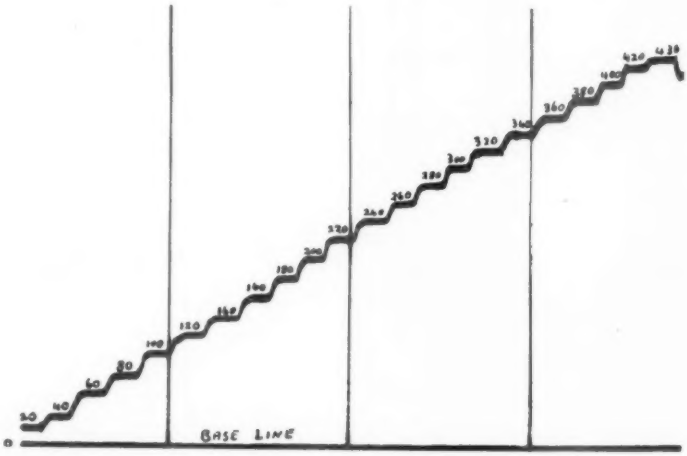


FIGURE IIB. Calibration tracing showing the base-line, the minute time markings and the effects of increments of pressure on the developed film. A rise of 100 millimetres of water in pressure causes a rise of 20 millimetres on the tracing. The end result is almost linear.

I shall now exhibit some of the tracings I have so far obtained.

The first is a normal tracing (Figure III) in which the intracranial tension runs a smooth course, whereas if it is increased by cerebral trauma or decreased by intravenous therapy as in these other films, it shows fluctuations in the record. In the same record is seen the Queckenstedt reaction—first by pressure on the right jugular, then on the left and then on both veins together. These are followed by a short series of coughs. It will

be noted that no matter how cerebro-spinal pressure is thus raised, whether rapidly by jugular compression or built up gradually as by coughing, the fall in either case runs parallel.

The next record (Figure IV) is similar to the foregoing. The pressure at the left of the record is 160 millimetres and then rises to 210 millimetres as a result of moderate pressure on the left jugular vein. Next is the effect

FIGURE III. Tracing taken from a normal person, showing how smoothly the pressure runs. The effects of jugular compression and of coughing are also shown.

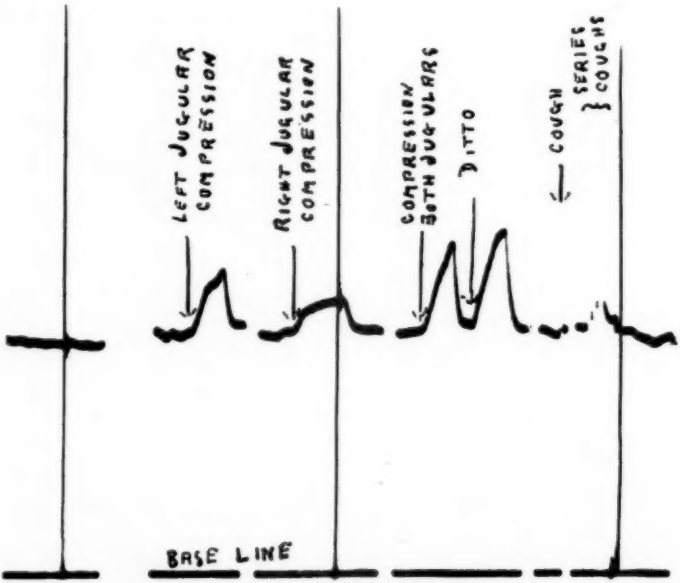


FIGURE IV. Tracing to show the effects of moderate jugular compression and coughing on the cerebro-spinal pressures.

of pressure on both jugulars together and finally the result of a cough and then a series of coughs.

The next tracing (Figure V) was taken after operation from a patient who had developed a secondary cerebral œdema. The initial pressure is 360 millimetres, and here we see in a pathological increase in pressure how much it fluctuates, how irregular it is and how much the cardiac and

respiratory factors of it are exaggerated. At the time shown, an intravenous injection of 50 cubic centimetres of 25% sucrose solution was given and a tracing over the next six minutes was taken, followed by a wait of a further fifteen minutes, during which the camera was stopped to see if anything further happened. Then I permitted cerebro-spinal fluid to escape until the pressure had dropped to 120 millimetres and took a further short tracing.

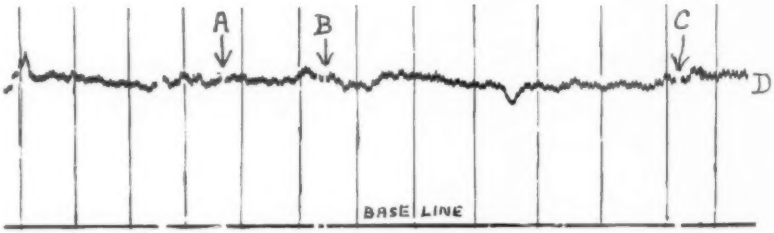


FIGURE V. Post-operative cerebral œdema, showing what little effect an injection of sucrose solution has in a case of raised cerebro-spinal pressure. This is followed by a lumbar decompression. A = intravenous injection of 25% solution of sucrose started, B = injection stopped, C = break in record for 15 minutes, D = pressure lowered 360 to 120 millimetres.

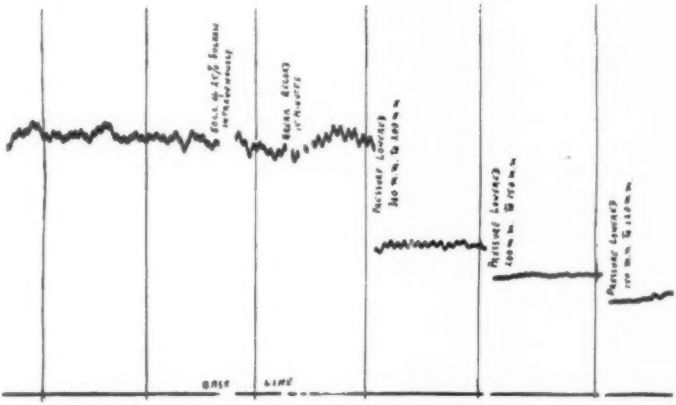


FIGURE VI. Post-operative cerebral œdema. Comparing the effects of an injection of hypertonic sugar solution with successive gradual spinal decompressions.

A review of this tracing shows that in a secondary cerebral œdema, sucrose solution in the amounts given does not appreciably affect intracranial tension, at any rate during a twenty-one minute period after it is injected, whereas decompression by the lumbar route is effective, more lasting and more nearly approaches a normal tracing in its form (compare Figure VI).

The final tracing I have to show again compares the effects of intravenous therapy with lumbar decompression in a case of post-operative cortical œdema.

The initial pressure was 300 millimetres and an injection of sucrose had but little effect on this, whereas successive gradual decompressions improved the form of the tracing and in the final reading at 120 millimetres it closely simulates a normal tracing (see Figure VI).

It is intended to prosecute future investigations to determine the differential effects of injections of 15% saline solution which, from the ionic concentration, that is, the larger number of small ions, might be expected to have a more rapid and more marked effect than glucose; but the latter has a nutritive value and its decompressive effect might be more prolonged.

Another important point that must be investigated, is the question as to whether intravenous hypertonic fluids produce a secondary œdema or a secondary increase of pressure, and, if so, how much.

I also intend making use of this machine to make a study of the effects of various commonly used anæsthetics on cerebro-spinal pressure over relatively prolonged periods of time. Such anæsthetics as ethyl ether, gas and oxygen, the intravenous anæsthetics and also divinyl ether will be tried, and a subsequent report on these findings will be forthcoming.

In conclusion, in reference to the data already to hand and from my own clinical experience, I have found that it is essential not to permit cerebral œdema to occur in acute cerebral trauma. I have also found that this can be prevented by keeping the intracranial tension at a normal level by lumbar puncture, and that even with blood-stained cerebro-spinal fluid, lumbar puncture is not contraindicated, since capillary oozing becomes less when the pressure is reduced to normal. In addition to the cortical damage that cerebral œdema can cause, it has also in my experience produced post-traumatic symptoms by the production of intracranial hypertension, the basis of which is probably some biochemical change associated with the œdema, not unlikely liberation of excess protein into the cerebro-spinal fluid with a consignment increase in the osmotic pressure.

INTRAPROSTATIC INJECTIONS.[1]

By Chas. M. Greenslade,
Dunedin.

Chronic prostatitis, whatever the cause, may prove resistant to orthodox methods of treatment. Injections into the prostate via the perineum (Figure I), as practised by Grant and Townsend, or transurethrally as recommended by McCarthy, are worthy of careful investigation. In 1917 Cano, Townsend and Valentine[1] introduced a method of treating the prostate by intravenous and intraprostatic injection of methyl phenol and normal phenol serum. Townsend,[2] who injected the prostate of a criminal, aged twenty-two years, five minutes after electrocution with a 20% suspension of ordinary indian ink in saline solution, has shown that substances injected into the prostate by perineal puncture diffuse throughout the gland. Also, since injected substances find their way into the prostatic blood vessels, no medicament should be introduced into the gland which will not be tolerated by the general circulation. Townsend has had considerable experience, having administered or supervised more than 1,000 intraprostatic injections. Grant[3] injects five to ten cubic centimetres of a 1% watery solution of mercurochrome into each lateral lobe of the gland. In 500 cases he has never had any cause to question the safety of the procedure; the clinical results have been almost universally satisfactory and in a great percentage almost phenomenal. In some cases the drug was undoubtedly injected outside the capsule without any deleterious effect. In 1933, while using Grant's method, I came to the conclusion that some of the injected solution must be escaping into the urethra, bladder or retroprostatic tissues; and so it was decided to inject the prostate with a substance opaque to X rays. "Uroselectan B" was chosen. Figure II shows the injection confined to the prostate. In Figure III the "Uroselectan" has extravasated along and beside the vesicles.

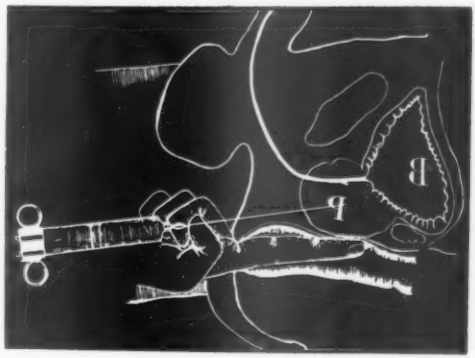


Figure I. Diagram of method of perineal intraprostatic injection. The finger in the rectum acts as a guide to the needle, and is used to disseminate the fluid throughout the gland by massage. As considerable pressure is often required, a Luer "Locktite" syringe fitted with an eight inch twenty gauge needle, as recommended by Grant, is used.

[1] Summary of a paper read at the annual meeting of the New Zealand Fellows of the Royal Australasian College of Surgeons on September 2, 1937. Accepted for publication on November 26, 1937.

This was proved by injecting cadavers wtih mercurochrome, when the coloured solution was found behind the bladder and beside and inside the vesicles as well as in the prostate. Figure IV shows a cystogram, the result of placing

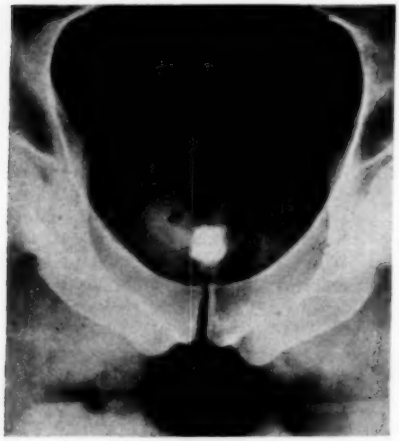


FIGURE II. Prostate injected with "Uroselectan B". The injection is confined within the capsule.

FIGURE III. The prostate injected with "Uroselectan B". The fluid has spread into and around the vesicles.

the injection into the posterior part of the urethra instead of into the gland. During injection a certain amount of mercurochrome usually leaks into the prostatic urethra, and is forthwith voided by the patient. In only one instance in a series of fifty cases was any serious ill effect noticed. In this instance the patient developed an acute cystitis a week after injection, the mercurochrome probably having been placed between the submucosa and muscularis of the bladder. Many patients showed definite improvement, and the injection seemed to mark the turning point in their progress towards cure. In other instances the improvement seemed to be mostly psychological. The mercurochrome stains the prostatic secretion for periods of as long as four weeks following injection.

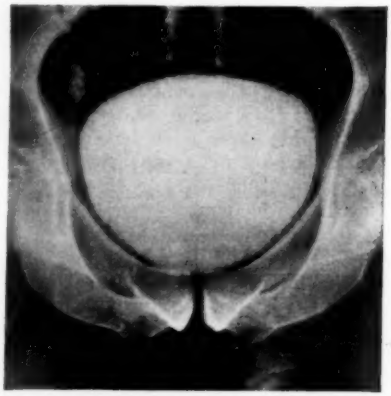


FIGURE IV. Attempted injection of prostate with "Uroselectan B". The needle probably pierced the prostatic urethra, hence the cystogram.

The McCarthy method,[4] in which "Electrargol" is directly injected by means of a long needle under vision either through the original panendoscope or as an attachment on the rack and pinion of the visualized electrotome, has also been tried with a 1% solution of mercurochrome instead of the

"Electrargol". It was found extremely difficult to inject more than a small portion of the gland at one sitting. McCarthy himself states that he has used the method in the treatment of forty patients, giving from three to seventeen treatments; all but two patients reported improvement. His article is a tribute to his personality, perseverance and patience, as well as to his patients' patience. Difficulties are encountered, especially in tough fibrous glands. The long fine needle, necessary to avoid damaging the prostate excessively, bends and is difficult to direct accurately. There is no trouble in placing the point of the needle in the gland, but how far in it should be pushed and with what pressure are problems not so easily decided.

Before injection therapy is used, it is well to clear up other foci of infection, such as those in teeth, gums, tonsils, sinuses *et cetera,* and to be certain that the lesion is not tuberculous, nor due to calculi. Tuberculosis of the prostate is usually associated with other forms of genital tuberculosis—of the epididymis and seminal vesicles—or with renal tuberculosis; and in most cases it is preceded by pulmonary phthisis, or at least by a history of pleurisy. Calculi can be excluded by X-ray examination.

Prior to injection in resistant cases of prostatitis, cysto-urethroscopy should be undertaken to exclude other causes of urinary shreds, such as stricture, retention from bar formation or diverticula, or again renal disease. A diagnosis of chronic prostatitis is sometimes made when the lesion is in the glands of Littré or Cowper, or in the vesicles. Cysts of the prostate, granulations, lymphoid bodies and polypi in the posterior part of the urethra are best removed by fulguration or with the endotherm knife. Prostatic sinuses and abscesses should be laid freely open through the endoscope. At the same time, double vasotomy is carried out in the presence of vesiculitis. In old-standing infections perineal intraprostatic injection is added, and is carried out after the endotherm knife has been used. This procedure must follow the endothermy, not precede it. The majority of patients with so-called incurable gonorrhœa, many of whom have had years of treatment, are cured by this means.

In an attempt to assess the ultimate results of intraprostatic injection, notice should be taken of the valuable work of O'Conor and Ladd,[5] whose experiments on dogs show the end results to be diffuse fibrosis of the gland. They state that any solution injected into the prostate, whether it is normal saline solution, distilled water, or of a so-called antiseptic character, results in destruction of the acini, chronic inflammatory changes and fibrosis.

The replacement of a gonorrhœal prostatitis with a fibrous prostate and subsequent bladder-neck obstruction may seem too high a price to pay for cure; but the urologist must look to the future and rely on his resectoscope or his punch to relieve this condition. In any event, many patients with uncured prostatic infections must end up with bladder-neck obstruction. Most men are willing to suffer a good deal of discomfort if they can be assured that they have been ridden of a gonorrhœal infection.

CONCLUSIONS.

1. Intraprostatic injections are of assistance in clearing up resistant prostatitis.

2. It must be realized that the end result of this therapy may be bladder-neck fibrosis, which in due course will need its appropriate treatment.

3. Most resistant cases of prostato-vesiculitis can be cured by endoscopic fulguration combined with vasotomy, with or without intraprostatic injections.

ADDENDUM.

During the period that has elapsed since this paper was read, mainly owing to the introduction of the sulphonamide compounds, the number of resistant cases of prostatitis has greatly diminished. With the widespread use of the sulphonamides in the treatment of gonorrhœa, there has been a marked decrease in the incidence of prostatic complications in this disease. In prostatitis itself, whether gonococcal or not, aside from tuberculous infections, the sulphonamide derivatives have proved of immense value in aiding resolution.

In addition, since using the method of Lowsley and Peterson for catheterization of the ejaculatory ducts,[6][7] I have noted the beneficial effects derived by the prostate as well as the vesicles.[8] In this technique phenol and glycerine in equal parts are applied through the Lowsley-Peterson endoscope to the *veru montanum* to relieve congestion, and to make the orifices of the ejaculatory ducts more easily seen. If applied to the *veru montanum* and prostatic fossettes, the phenol and glycerine make the orifices of the ejaculatory ducts and prostatic ducts gape open. This would appear to encourage free drainage of the glands, and thus aid in resolution of the vesiculitis and prostatitis. As a direct result of these advances in therapy, the indications for intraprostatic injections in prostatitis have practically disappeared.

REFERENCES.

[1] F. G. Cano, T. M. Townsend and J. J. Valentine: "Intravenous and Intraprostatic Injections of Methyl Phenol and Normal Phenol Serum in Gonorrhœa", *Medical Record*, April 28, 1917, page 715.

[2] T. M. Townsend: "Intraprostatic Injections", *Journal of Urology*, Volume xxxv, 1936, page 75.

[3] O. Grant: "The Treatment of Chronic Prostatitis by Injection", *Journal of Urology*, Volume xxxiii, 1935, page 631; *ibidem*, Volume xxix, 1933, page 749.

[4] J. F. McCarthy: "Recent Advances in Instrumental Urology", *Journal of Urology*, Volume xxxiii, 1935, page 303.

[5] V. J. O'Conor and R. L. Ladd: "Intraprostatic Injection: An Experimental Study", *The Journal of the American Medical Association*, Volume cvii, October 10, 1936, page 1185; *Journal of Urology*, Volume xxxvii, April, 1937, page 557.

[6] O. S. Lowsley and A. P. Peterson: "The Lowsley-Peterson Universal Endoscope: A New Instrument and its Uses", *The American Journal of Surgery*, Volume xxxiv, 1938, page 168.

[7] A. P. Peterson: "Retrograde Catheterisation in Diagnosis and Treatment of Seminal Vesiculitis", *The Journal of Urology*, Volume xxxix, May, 1938, page 662.

[8] C. M. Greenslade: "Catheterisation of the Ejaculatory Ducts", *New Zealand Medical Journal*, Volume xxxix, April, 1940, page 88.

PORTAL PYÆMIA FOLLOWING ACUTE APPENDICITIS: CASE OF MULTIPLE LIVER ABSCESSES WITH RECOVERY.[1]

By Russell N. Howard.

The successful outcome of the following case of portal pyæmia with multiple liver abscesses seems worthy of note, since it is generally believed to be, in its more acute forms, a universally fatal condition. An extensive perusal of the literature showed that, although this belief is incorrect, an additional solitary recovery would take a welcome place in medical records.

Further, as this search revealed that nothing approaching a complete exposition of the disease had been written, I have taken the opportunity of recording a general description of the malady and its treatment. I do not pretend that this is exhaustive, but it embodies an account of the condition as a complication of appendicitis and a more comprehensive estimate of the number of cases of recovery than has yet to my knowledge been published.

Case Report.

D.W., a male, aged seven years, was admitted to Saint James's Hospital, London, S.W.12, on October 21, 1935, with a sixty-hour history of acute appendicitis presenting no unusual features. His temperature was 101·4° F., his pulse rate was 116 and his respirations numbered 20 per minute. The lower part of his abdomen was tender and rigid.

The first operation was performed on October 21, 1935. Under ether anæsthesia, a muscle-splitting incision was made and a gangrenous retrocaecal appendix was removed. Lower abdominal peritonitis, with much purulent fluid, was present. A drainage tube was placed in the pelvis through a suprapubic stab incision and the wound was closed, with drainage of the subcutaneous tissues.

Both drainage tubes were removed in forty-eight hours. Gross wound infection developed. The temperature swung to 101° F. at night for the next two weeks and during the third week subsided by lysis. After a further week, the temperature rose again and became irregularly remittent and intermittent, occasionally as high as 101° F., for the next fortnight. This was followed by a week of normal temperature without clinical improvement.

The wounds were now cleaned and healed, but the abdominal wall felt thin on palpation over the iliac fossa wound. The patient was pallid and listless and the hair of the head had begun to fall out. Examination revealed some upper abdominal tenderness.

On December 4, 1935, the patient complained of chilly feelings for the first time, although the temperature was now normal. On December 8, 1935, the temperature began to rise, until, showing morning intermissions, on December 13, 1935, it reached 102° F. in the evening. On this date further chills were experienced.

Examination revealed fullness and tenderness in the upper part of the abdomen and dulness on percussion, with diminished breath sounds and egophony at the base of the right lung. X ray examination—by screen and film—revealed uniform elevation of the right half of the diaphragm, with diminished respiratory excursion.

A probable right posterior intraperitoneal subphrenic abscess was diagnosed, but as the temperature proceeded to fall by lysis, and some clinical improvement occurred,

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operation was deferred despite a leucocyte count of 35,000 per cubic millimetre. The patient's condition at this time was poor; he was very pallid, had lost much weight, was practically hairless and had almost complete anorexia. An irritating dry cough had developed.

The temperature again became irregularly remittent, with an evening maximum of 102·5° F. The edge of the liver was felt one hand's breadth below the right costal margin, and the spleen was easily palpable. X ray examination revealed further elevation of the diaphragm.

The second operation was performed on December 26, 1935. Under gas and oxygen anæsthesia, a transverse muscle-cutting incision was made at about the tip of the eleventh rib. Two ounces of foul pus were evacuated from the right posterior intraperitoneal space; the right anterior intraperitoneal space appeared free of infection. The liver was enlarged and its surface appeared normal. A drainage tube was inserted into the abscess cavity.

This was followed by subsidence of the temperature to 99° F. or 100° F. in the evening, with an occasional higher peak. No improvement in the patient's condition, however, occurred. The liver proceeded to enlarge steadily and came to occupy most of the upper part of the abdomen and, on the right side, some of the lower part.

His general condition now became desperate; ascites developed and an X ray examination showed further elevation and complete immobility of the right side of the diaphragm. The leucocyte count was 46,000 per cubic millimetre.

Suppurative pylephlebitis with intrahepatic abscess formation was diagnosed and operation again resorted to.

The third operation was performed on January 17, 1936. Under gas and oxygen anæsthesia, supplemented with local infiltration with 1% "Novocain" solution, the subphrenic area was needled through the lower right intercostal spaces posteriorly, but no pus was obtained. Two inches of the tenth rib were resected in the scapular line and the pleura opened in the phrenico-costal sinus. This contained a large amount of serous fluid, which was evacuated. The diaphragm was then sutured to the pleura, incised across its fibres and the liver exposed. No pus was seen, and none was obtainable by digital exploration. The right lobe of the liver appeared normal, but was needled. Bile-stained pus was obtained at a depth of one and a half inches. A scalpel was passed beside the needle and followed by the finger, which entered an abscess cavity the size of a tennis ball. A partition was broken down and a further smaller cavity was opened. Much bile-stained pus welled out. A rubber drainage tube was inserted and the operation concluded.

During the next few days considerable clinical improvement occurred, with subsidence of the temperature, except for one further peak on the twelfth day following this procedure.

A biliary fistula, complete, as evidenced by quite clay-coloured stools, now developed through the drainage tube. The tube was removed on the seventh day, but the fistula persisted. The patient's bile was collected and fed to him intraduodenally with a Ryle's tube.

This state of affairs was maintained until March 11, 1936 (that is, seven weeks after the third operation), when the fistula closed spontaneously and quite suddenly. All this time the liver had remained very large and ascites was still present. A definite bulge was present in the iliac fossa scar.

An X ray film, taken with the patient in the erect posture, on February 17, 1936, had shown a small fluid level posterior to the xiphisternum; this seemed to be due to a small abscess in the liver, the size of a golf ball, containing fluid and gas. The temperature and pulse rate were now normal and the patient was apparently well, but it was thought advisable (in retrospect, probably wrongly) to open this abscess.

The fourth operation was performed on February 24, 1936. A right upper paramedian incision was made beside the xiphisternum and the liver was exposed. Much clear ascitic fluid escaped from the peritoneal cavity. Both right and left lobes of the liver were needled extensively without pus being found. The surface of the liver showed many projecting firm masses the size of a hazel nut. These were considered to be inspissated fibrosing abscesses and were not interfered with. Although the abscess sought in the liver was not found, no intraperitoneal condition capable of explaining the radiographic findings was discovered. The abdomen was then closed.

The temperature rose again on March 4, 1936, and showed slight elevation until March 15, 1936, when it fell to normal and remained so until the patient left hospital.

During this time the liver had begun to recede and the ascites to diminish. The spleen was now not palpable.

At the same time the hair began to grow, and the patient was discharged from hospital on May 16, 1936 (eighty-one days after the fourth operation and two hundred and eight days after admission).

When examined on September 21, 1936, he was apparently a normal child. The liver and the spleen were not palpable and the bulge over the iliac fossa wound was not apparent. Hair was present on the scalp in normal amount.

Comments.

1. This case is one of portal pyæmia of the acute type, with liver abscess formation.

2. The case is characteristic, except that chills were inconspicuous and jaundice was absent.

3. The development of the biliary fistula and its maintenance for two months are unusual. The fact that the pus was bile-stained indicates erosion of an intrahepatic bile duct and this was apparently a large one. As most similar cases recorded in the literature were characterized by some degree of jaundice, it is possible that, but for the development of the fistula, jaundice would have occurred in this patient.

4. Looked at in retrospect, the fourth operation was undoubtedly unnecessary, but served to demonstrate the curious pathological feature of inspissated abscesses projecting from the liver surface. This will be referred to later.

PATHOLOGY.

Incidence.

Portal pyæmia occurs in about 0·4% of cases of acute appendicitis. It has been variously estimated from 0·13% by Snyder, Hall and Allen[71] to 1·33% by Braun[12] (see Table I).

TABLE I.

Incidence of Portal Pyæmia.

Author.	Total Cases.	Cases of Pyæmia.	Percentage.
Eliason[29]	2,237	3	0·13
Stewart-Wallace[74]	5,471	8	0·15
Koster and Kasman[47]	1,027	3	0·29
Snyder, Hall and Allen[71]	8,969	27	0·30
Colp[27]	2,841	9	0·32
Short[70]	1,000	4	0·40
Petrén[63]	2,779	15	0·54
Brütt[17]	2,500	15	0·60
Gerster[26]	1,189	9	0·76
Braun[12]	600	8	1·33

Condition of the Appendix.

The appendix is generally the site of an acute gangrenous inflammation, frequently with local abscess formation. Rolleston and McNee[68] suggest that pus under pressure is a predisposing cause. However, all degrees and stages of acute and chronic inflammation may be found in different cases. The appendix rarely may appear macroscopically normal. In one such case a pure culture of hæmolytic streptococci was grown from the appendix and the

same organism was isolated, in pure culture, from a thrombus in the ileo-colic vein.

It has been wrongly stated by Brütt,[17] Colp[27] and others that in cases in which appendicectomy is performed within twenty-four hours of the onset of symptoms, portal pyæmia does not develop. Actually, in such cases, portal pyæmia is a rare, but not an unknown, complication.[29][74] Apart from this, it does not seem to be more common in cases which have not been operated on.

Pathology of Spread to Liver.

The spread is by the ileo-colic vein—perhaps occasionally by the lymphatics about it. The mesoappendix is œdematous and friable and its veins are thrombosed. Thence the spread may proceed in the vein by: (*a*) Embolism. (*b*) Progressive mural thrombosis: the blood current in the central part of the vein is uninterrupted; the vein feels thickened and the tissues about it are œdematous. (*c*) Progressive complete thrombosis: suppuration is usually present at intervals throughout the thrombus and may involve it in its whole length. It may spread by direct continuity through the superior mesenteric and portal veins to the liver and thence along the finer radicles into the liver lobules. Retrograde thrombosis may occur along the splenic vein and splenic abscess may result. Thrombosis may occur in the hepatic veins and emboli may then lodge in the lungs with the formation of pulmonary abscesses.

Suppuration may extend beyond the walls of the veins with the formation of abscesses along their course. There is gross œdema of the peritoneum and retroperitoneal tissues, and, if the superior mesenteric vein is involved, of the mesentery.

Changes in the Liver.

Macroscopically the liver is enlarged. Small yellowish dots of suppuration may be seen on the surface and larger abscesses may produce localized bulgings. The cut surface shows a fatty appearance with abscesses of various sizes, varying in number and size within wide limits in different cases. In the acute cases the abscesses are generally multiple and in both lobes. In the subacute and chronic cases there may be one large solitary abscess, generally in the right lobe, but sometimes in the left. The abscesses contain pus of varying thickness which may be bile-stained (should erosion of a bile duct have occurred). Pus may burst through the diaphragm or into the peritoneum or extraperitoneal tissues and occasionally into adjacent viscera, for example, the stomach. As the abscess ages, the pus increases in thickness and the wall becomes harder. Eventually it may appear a firm homogeneous mass which may bulge from the liver surface. The case here recorded presented such a condition at the fourth operation.

Microscopically, thrombus formation can be seen in the portal vein radicles, with degenerated liver cells about the thrombi. Later, abscess formation occurs here. In the acute cases the intact liver cells show a cloudy swelling and fatty degeneration. As mentioned above, thrombosis may be present in the hepatic veins.

Should recovery occur, the liver may return to normal size, but fibrosis and scarring will be present.

Associated Lesions in the Abdomen.

All varieties of subphrenic abscess may be present, generally arising more directly from the appendix, but sometimes owing to extension of suppuration from the veins or to the rupture of a liver abscess. Other intraperitoneal abscesses are not unusual and occasionally rupture of such abscesses or of a collection in the liver may cause a general peritonitis.

Splenic enlargement is common and splenic abscess may occur. Ascites is not infrequent in the later stages.

Thoracic Lesions.

Some degree of right pleural effusion is usual. Empyema may occur should this effusion become infected, with or without rupture of a liver abscess through the diaphragm. In the more chronic cases with large liver collections, one may burst through adhesions into the lung and the contents be coughed up. Lung abscesses may occur in both lungs owing to embolism from hepatic veins in acute cases. Pneumonia may be present independently of these occurrences. All organs show evidence of toxæmia.

Bacteriology.

As one would expect from the known bacteriology of acute appendicitis, the organisms most frequently concerned in clot and abscess formation are *Bacillus coli communis*, streptococci, and anaerobes; the pneumococcus has been recorded. They are to be found in the clot, clot abscesses and liver abscesses, and have been obtained on blood culture in some of the cases in which such examination has been made.

CLINICAL FEATURES.

The clinical features may be differentiated into three groups:

1. *Features Associated with the Primary Cause (Appendicitis).*—In the hyperacute case it may be difficult to dissociate the clinical features from the symptoms of portal pyæmia, as the onset of this may occur within twenty-four hours of the onset of the acute appendicitis and thereafter a composite clinical picture be presented. In the subacute and chronic types of case the history of the primary cause may be concealed in the obfuscations of the patient's memory. In most cases, however, the appendiceal attack is recent and severe; the patient may or may not have been subjected to appendicectomy.

2. *Evidences of Intravascular Sepsis.*—The onset of this phase, that is, portal pyæmia, is generally with a rigor, which is perhaps its most characteristic feature. Rigors then occur two or three times in the twenty-four hours. Chilly feelings may be complained of without any actual rigor, as in the case reported here. Cases not exhibiting this feature are extremely rare. At the same time the temperature is elevated, becoming irregularly remittent or intermittent, with peaks up to 103° F. or 105° F. in the evenings. Clinical evidences of toxæmia appear, as in other acute febrile illnesses.

Abdominal pain is almost invariable, and is associated with abdominal tenderness and rigidity. These characters are right-sided, over the region of the caecum, ascending colon and liver. Congestion of that part of the bowel, the venous return from which passes along the ileo-colic vein, is probably

responsible for the pain, the congestion being caused by thrombosis of this vein. Referable to the same condition is the occasional occurrence of the passage of dark blood by the bowel, sometimes in amounts up to three or four ounces. Vomiting is quite common, and diarrhœa of the septic type may occur in the later stages.

3. *Indications of Liver Involvement.*—Liver enlargement is invariable, is usually both upwards and downwards, and may attain considerable dimensions, as in the case recorded above, in which the liver seemed to fill most of the abdomen. The enlargement begins shortly after the onset of rigors—within twenty-four hours—and may be associated with jaundice. Actually jaundice appears in about 50% of cases. Its absence in my case and the possible reason therefor (biliary fistula) have already been commented on. Eliason[29] mentions œdema over the liver as being a feature, but this probably occurs only in association with a subphrenic abscess. Enlargement of the spleen is present in about 50% of cases and may be associated with splenic abscess. It was present in the case under consideration, but without apparent abscess formation.

Referable to the upward enlargement of the liver is the development of chest features. Signs of elevation of the right diaphragm and compression of the base of the right lung are practically invariable—dulness on percussion, diminished breath sounds, egophony and perhaps distant tubular breathing. A sympathetic right pleural effusion will aid the production of these signs. They are out of all proportion to the respiratory symptoms, which are usually represented by a cough. This may be irritating or quite inconspicuous. In my case the signs were very gross and the cough was irritating.

Later in the course of the disease ascites and enlarged veins in the abdominal wall may develop. These features are unusual, but were very prominent in the later stages of my case. They disappear completely should recovery occur.

Special Features.

The leucocyte count varies considerably. Koster and Kasman[47] report cases with a count of 12,000 and 13,000 per cubic millimetre. In Cabot Case 18472[22] there was a count of 45,000 per cubic millimetre. The patient reported in this article had a higher count (46,000 per cubic millimetre) than any I can find recorded. With regard to blood culture, comparatively few findings are mentioned. Brütt[17] remarks that culture is useful, yielding anaerobic streptococci and *Bacillus coli communis.* Colp[27] and Eliason,[29] on the other hand, say attempts at culture are generally unsuccessful. The commonest organism yielded on culture is undoubtedly *Bacillus coli communis.* The hæmolytic aerobic streptococcus is next most common.

The radiographic features are important. Under the fluorescent screen the right half of the diaphragm is seen to be raised and its excursion much diminished. This is almost invariable. The left half of the diaphragm may be raised slightly, but its excursion is less affected. An X ray film confirms this, and if taken with the patient in the erect posture may, rarely, show a fluid level in the liver, as in my case.

Clinical Types of Case.

Hyperacute Type.—The hyperacute type complicates a case of acute appendicitis. The onset is soon after that of the primary disease—usually

within the first three to five days. If the onset is delayed longer, the patient will probably have shown signs of continued and perhaps progressive infection; appendicectomy may or may not have been performed. Sometimes, as has been mentioned above, the onset of portal pyæmia is so linked with the onset of appendicitis that it is difficult to separate the two sets of clinical features. This hyperacute case has its onset with a rigor, rapid elevation of temperature, abdominal pain, vomiting, further rigors, jaundice, enlarged tender liver, probably an enlarged spleen and a rapid septic course. Death within a few days is almost invariable.

Acute Type.—The acute type also complicates acute appendicitis. The onset is longer delayed, even up to six weeks after the onset of the primary disease. Again the appendix may or may not have been removed, and the patient may have shown signs of lack of subsidence of the infection in some degree. Wound infections and intraperitoneal abscesses are common. In this type, the onset is with a rigor, rapid elevation of temperature and the development of the signs detailed above, but the course is not so rapid, not so severely toxic. The survival period in these cases is measured rather in weeks than days, although many patients die in the second week. However, the outlook is not hopeless and every endeavour must be made to conduct treatment so as to allow the best chance of recovery. Liver abscesses are usually multiple, but may be solitary.

Subacute and Chronic Types.—Subacute and chronic types may complicate acute appendicitis, occurring weeks, months or even years after subsidence of an attack or after successful appendicectomy. They have been said to occur also during the course of chronic appendicitis. The onset is much more insidious and the course prolonged. Malaise, chills, rise in temperature and later liver enlargement, both upwards and downwards, occur. Hepatic pain is present. The leucocyte count is elevated and the picture is that of hidden pus. Jaundice may be present, splenic enlargement is rare and ascites absent. The diagnosis is confirmed at operation and a large solitary abscess, usually of the right lobe, is found. These cases have a much higher recovery rate than the former varieties.

Other Types.—The description of clinical types is not complete without mention of two other types of condition which are not strictly speaking portal pyæmia, but which almost certainly represent on the one hand a milder and on the other a more severe grade of the same process. Attention to this fact is drawn by Reök.[65]

The first of these is mild toxic jaundice. Such cases are not very uncommon, are benign, and respond rapidly to administration of glucose. They present a mild toxæmia, jaundice and hepatic enlargement, occurring early in the course of appendicitis or shortly after appendicectomy. They are not associated with rigors.

The second of these other types is acute fatal hepatitis. This is a fulminating toxæmia, invariably fatal and of rare occurrence. There are no rigors, the leucocyte count is normal or subnormal, and death occurs within forty-eight hours.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

Acute and Hyperacute Cases.—Diagnosis of the fully developed case of portal pyæmia offers few difficulties. The time to make the diagnosis, however,

is in the early stages and, if possible, before the advent of liver involvement—certainly before spreading clot has involved the portal vein. At this stage one can still make an attempt to prevent abscess formation in the liver.

Early diagnosis is to be made on three features. Rigor or chill without other obvious cause is the most significant symptom. This is accompanied by rapid rise in temperature and usually by right-sided abdominal pain.

The initial symptom is, in practically every case, a rigor or chill. Chills in appendicitis occur in a variously estimated percentage of cases. Snyder, Hall and Allen[71] estimate it at 2·1% (8,969 cases), Colp[27] at 6·8% (2,841 cases) and Kelly[43] at 15%. As the percentage of patients developing portal pyæmia is approximately 0·4, it is obvious that all patients with chills have not developed this complication. The rigors in portal pyæmia, however, tend to be repeated, and after the third rigor I consider that the abdomen should be opened, the diagnosis made beyond question and early operative treatment instituted (*vide infra*).

A positive blood culture at this stage would clinch the pre-operative diagnosis; a "negative" one has no significance.

In the differential diagnosis pyelitis will have to be excluded by urinary examination and the absence of renal tenderness.

Subphrenic abscess, pelvic abscess and other intraperitoneal and wound abscesses may be a source of error. Frequently, however, the onset is too early for the development of these conditions, which must be excluded by the local examinations adapted to their situation. Subphrenic abscess is the most difficult, mainly in those cases of portal pyæmia not characterized by repeated rigors (and, further, one must also bear in mind that the two conditions may be associated). Should jaundice develop, the diagnosis is reasonably certain. Without jaundice an X ray examination may be, but is usually not, conclusive, and the diagnosis will then be made beyond doubt at operation.

Disease of the biliary tract is not liable to cause diagnostic difficulty in cases which are known to be primarily appendiceal, that is, post-operative cases and most cases in which operation is not performed. Others will require consideration from the point of view of age, sex, bodily habitus, and above all an accurate history of present illness and past attacks. The last mentioned is frequently of greater aid than physical examination, with which it must be correlated.

General systemic pyæmia, as a complication of appendicitis, is very rare. The differential diagnosis, in the later stages, is made by the absence of signs of liver involvement in this condition. In the very early stages exact differential diagnosis is impossible.

Right diaphragmatic pleurisy, pneumonia and empyema are eliminated by the absence of repeated rigors in these conditions, their own peculiar physical signs and X ray examination.

This completes the early diagnosis of the acute and hyperacute cases. Later diagnosis is relatively easy and is made by a full consideration of the features detailed above.

Subacute or Chronic Cases.—Subacute and chronic cases offer quite a different diagnostic problem. Although border-line cases occur which are difficult to pigeon-hole, there is practically never any question of diagnosis, and probably never of operation, before the advent of liver involvement.

Following some time after a successful appendicectomy, after a subsided attack of acute appendicitis (the history of which may be difficult to elicit), or during the course of chronic appendicitis (with an even more difficult history to evaluate) comes an illness with the features of internal suppuration and later with signs referable to the liver region. In these cases there are the large solitary abscesses with a very subacute or chronic course as detailed earlier.

Diagnosis is made firstly on the features of internal suppuration, secondly on signs implicating the right upper quadrant of the abdomen, thirdly by the exclusion of other conditions, and fourthly by operation.

PROGNOSIS.

It is my purpose in this article to emphasize the fact that the prognosis of this condition, although grave, is not completely hopeless. One cannot give even an approximate percentage of recoveries because, firstly, successful cases are usually reported, whereas those which are unsuccessful generally remain in obscurity, and, secondly, certain conditions, reported as liver abscesses, appear from the case histories to have been subphrenic abscesses only. In the compilation of Table IV these have been excluded, and the criterion of liver abscess has been at least one of the following: an account of the exploration of the liver with discovery of pus as a distinct operative step; a definite statement from the author that the abscess cavity was felt to be in the liver; the finding of bile-stained pus at the first operation on the liver region.

The subacute and chronic cases (as defined earlier) will be dismissed first with the statement that the recovery rate in those which are accurately diagnosed and in which adequate treatment is carried out is comparatively high—probably in the neighbourhood of 50%.

This article is more concerned with the hyperacute and acute cases, and these I shall now proceed to discuss.

Firstly, with regard to the possibility of spontaneous recovery: this may undoubtedly occur, as exemplified by a typical clinical case of Sir Frederick Treves[79] in which, at operation, minute abscesses were found dotted over the liver surface and in which recovery occurred after closure of the abdomen without any local treatment. As operative interference had failed to accomplish any immediate objective, the patient would probably have survived without it. Lilienthal[50] reports a similar case, except that abscesses were discovered only after microscopy of a biopsy specimen; this patient also recovered. In many cases, including my own, in which the abdomen has been opened and abscess cavities have been drained, recovery has occurred, despite the fact that abscesses in the liver, recognized at operation, were not interfered with. Finally, there are cases with typical clinical features in which recovery has taken place without operative intervention. These cases cannot be diagnosed with certainty, but they furnish a further link in the chain of evidence for spontaneous recovery.

When we come to consider operative recovery, we rest on more certain ground. Surgical procedures are discussed later, but here one can say that amongst the early operations, that is, primary and secondary venous ligations, fifteen undoubted successes have been reported (see Tables II and III). Of

these, thirteen operations were primary ligature, and in this number Braeunig's[11] four patients and Sudeck's[75] one patient recovered so dramatically as to suggest that recovery was very probably determined by the surgical measures adopted. The poor results of secondary venous ligation are probably due to the late time of operation. Operative interference, then, in these cases seems to hold out hope of preventing liver abscess formation, and, if resorted to sufficiently early, should improve the prognosis.

TABLE II.

Cases of Primary Venous Ligation.

Author.	Vein Ligated.	Recoveries.	Deaths.	Total Number of Cases.
Braeunig[11]	Ileo-colic.	4	1	5
Braun[12]	Ileo-colic.	2	—	2
Brunner[16]	Ileo-colic.	1	—	1
Brütt[17]	Ileo-colic.	1	—	1
Fromme[34]	Ileo-colic.	1	—	1
Hempel[39]	Ileo-colic.	1	2	3
Reök[63]	Ileo-colic.	1	—	1
Sudeck[75]	Superior mesenteric.	1	—	1
Weil[82]	Ileo-colic.	1	—	1
Total		13	3	16

TABLE III.

Cases of Secondary Venous Ligation.

Author.	Vein Ligated.	Recoveries.	Deaths.	Total Number of Cases.
Beer[6]	Portal.	—	1	1
Brütt[17]	Ileo-colic.	—	2	2
Colp[27]	1 Superior mesenteric. 3 Portal.	—	4	4
Kleinschmidt[43]	Ileo-colic.	—	1	1
Melchior[53]	Ileo-colic.	1	—	1
Stewart-Wallace[74]	Superior mesenteric.	1	—	1
Total		2	8	10

Late operation is concerned with the treatment of hepatic abscess. Forty-two recoveries in which liver abscesses were undoubtedly demonstrated have been discovered in the literature (see Table IV), and the case reported here brings the total to forty-three. It is significant that in thirty-one of these cases solitary abscesses were present. In addition to these, one recovery is attributed to both Chvostek[24] and Verebély,[80] but I have been unable to obtain these case reports. The case of Schiltz[69] was not a proved example of liver abscess, although probably one of portal pyæmia.

With regard to the data on which a prognosis may be formed, the following broad principles are all that can be laid down: firstly, the prognosis is always grave; secondly, the longer the onset of symptoms after the primary appendicitis, the better the outlook; thirdly, the less rapid the progress of

the disease, the more likely is recovery to occur; fourthly, abscesses, apparently solitary at operation, have a more favourable outlook than those which are obviously multiple.

TABLE IV.

Cases of Liver Abscess with Recovery.

Author.	Multiple Abscesses.	Solitary Abscess.	Total.
Barnes and Pearson[5]	1	—	1
Bidwell[7]	—	1	1
Bitner (case summary by Petrén)[8]	—	1	1
Brogden[13]	1	—	1
Bruggeman[15]	1	—	1
Clarke[25]	—	2	2
Ellason[30]	1	5	6
Elsberg[30]	—	1	1
Franke[33]	—	1	1
George[35]	—	1	1
Halstead[37]	—	1	1
Hohlbaum[40]	—	1	1
Holden and Moran[41]	1	—	1
Jalaguier[43]	—	2	2
Kelly[45]	—	1	1
Körte[46]	1	1	2
Lepetit[49]	—	1	1
Lilienthal[50]	1	—	1
Loison[51]	—	1	1
Makrowski (case summary by Petrén)[53]	—	1	1
Mgaloblischwli (case summary by Walter-Sallis)[54]	—	1	1
Morton[55]	1	—	1
Moschowitz[56] [57]	—	1	1
Munro[58]	—	1	1
Perman (case summary by Petrén)[59]	—	1	1
Petrén[60]	—	1	1
Quénu and Mathieu[64]	—	1	1
Rocher[67]	—	1	1
Snyder, Hall and Allen[71]	—	1	1
Soligoux and Roederer (case summary by Petrén)[72]	—	1	1
Syms[74]	1	—	1
Thyne[78]	—	1	1
Treves[79]	1	—	1
Weil[82]	1	—	1
Total	11	31	42

PROPHYLACTIC TREATMENT.

Prophylaxis is directed towards one object—appendicectomy within twenty-four hours of the onset of acute appendicitis. Portal pyæmia, although not unknown in such operative cases,[29][74] is of extremely rare occurrence. Frequently this goal is impossible of attainment, owing to the vagaries of the patient, but this much maligned character is not always to blame.

As the details of diagnosis of acute appendicitis can hardly be discussed here, I propose to leave this point with a plea that, when diagnosis has been made within the first forty-eight hours, no consideration be allowed to stand in the way of immediate operation.

There is a type of case in which the patient, who is first seen at a period more than forty-eight hours from the time of onset, has a mass in the abdomen and the condition shows signs of subsidence. Such patients are frequently treated along Ochsner-Sherren lines with excellent results, provided that all the dictates of this treatment are adhered to. There does not appear to be any increased incidence of portal pyæmia as a result.

TREATMENT OF THE ESTABLISHED CONDITION.

Chemotherapy.

Chemotherapy, in its general application to medicine and surgery, has recently received a most justifiable impetus. Isolated cases of probable portal pyæmia in which the patient has recovered after the intravenous exhibition of eusol and mercurochrome have been recorded.[3][41] Judging by the lack of favourable results with such solutions in comparable conditions (for example, puerperal septicæmia), one is doubtful of their real efficacy in these cases. Such recoveries are probably *post hoc* but not *propter hoc*. Sulphonamide preparations are in a quite different category. The organisms concerned have been proved susceptible to attack by these drugs, and their use may well be of the greatest value. General supportive treatment will, of course, be adopted in routine fashion.

Operative Treatment.

Venous Ligation.—Venous ligation (see Tables II and III) may be primary—that is, ligation at the primary surgical attack on the pathological process, when appendicectomy will probably be performed also—or secondary —that is, performed at a subsequent operation.

Most patients suffering from portal pyæmia die because of affection of the liver, usually with abscess formation. Infection almost invariably passes to the liver along, successively, the veins of the mesoappendix, veins passing over the caecum, veins of the ileo-caecal angle, ileo-colic, superior mesenteric and portal veins. If, in the early stages, this venous system can be ligated proximal to the infection, the liver may escape gross involvement. Many cases of such ligation are on record. With regard to the level of ligature, it is essential that it should be proximal to any pathological condition in the vein, provided that this can be done without the certainty of causing the patient's death.

Portal Vein Ligation. From the experimental side it has been shown by Boyce, Lampert and McFetridge,[10] and again by Neuhof,[59] that ligation in one stage of the portal vein of healthy dogs is incompatible with life; it was, however, able to be done successfully in three stages. Further, Wilms[85] ligated the portal vein in a case of penetrating injury, and the patient survived. This appears to be the only case in which a successful portal vein ligation has been recorded in the human subject. In the operative therapy of portal pyæmia, Beer[6] has once, and Colp[27] three times, practised portal vein ligation. All patients died, despite the fact that one of Colp's operations was done in stages and the patient lived for more than two weeks. It is of interest, however, that, as shown by *post mortem* examination, none of the patients developed intestinal gangrene. Thus portal vein ligation does not offer much hope in the treatment of this condition.

Superior Mesenteric Vein Ligation. Superior mesenteric vein ligation has twice been done successfully[74][75] for portal pyæmia, and at least once[27] has been followed by death. Further, the superior mesenteric vein has once been ligated successfully in a case of perforating abdominal injury.[66] The procedure, therefore, is compatible with life, provided that one or more jejunal veins join the superior mesenteric proximal to the ligature to allow venous drainage of the small intestine.

Ileo-Colic Vein Ligation. A number of cases of successful ligation of the ileo-colic vein are on record. The operation was first recommended by Braun,[12] two of whose three patients recovered. Braeunig[11] among five primary ligations had four recoveries. Brütt[17] had one successful primary ligation and two unsuccessful secondary ligations. Hempel,[38] in three primary ligations, had one recovery. Brunner,[16] Fromme,[34] Reök[65] and Weil[82] have each accomplished one successful primary ligation, and Melchior[53] a successful secondary ligation. Many of these patients showed a dramatic improvement immediately after vein ligation. Others proceeded in such a way as to suggest that this procedure had little real effect on the course of the disease. The results are sufficiently encouraging, however, to make one believe that a ligature applied proximal to the clot has a very definite value in preventing liver infection.

Ileo-Colic Angle Vein Ligation. Wilms[84] has recorded recovery of a patient who had symptoms suggestive of portal pyæmia, and who was treated by ligation of the ileo-colic angle veins. The operation is almost necessarily incomplete, however (to ligate all the veins, and only the veins, in a thick œdematous ileo-colic angle is difficult). Furthermore, Sprengel[73] has reported a case in which a patient, treated thus, developed caecal gangrene and died with hepatic abscesses.

Mesoappendix Vein Ligation. Gerster[36] advocated the ligature and extirpation of the veins of the mesoappendix, along with the veins passing over the caecum. He reported two successful cases, but himself admitted that usually the operation was not practicable. It is obviously subject to objections similar to those associated with Wilms's operation.

In conclusion, the most suitable operation appears to be that of Braun[12] —ligature of the ileo-colic vein close to its entrance into the superior mesenteric. If clot extends beyond this point, the superior mesenteric vein itself may be ligated, provided that some jejunal veins are left proximal to the ligature. One advocates this procedure in cases in which, at a primary or secondary operation, clot is felt in the ileo-colic vein, or in which gross œdema of the peritoneum and tissues about the vein makes its occlusion a certainty. In cases of acute appendicitis with suspicious symptoms the presence or absence of these features should be expressly ascertained, and they should be present before vein ligation is attempted. A friable, non-bleeding mesoappendix is not uncommon, but does not, for this reason alone, justify such procedures. Portal vein ligation is never recommended.

TREATMENT OF ESTABLISHED LIVER SUPPURATION.

In the hyperacute case the patient does not survive to reach the stage at which local treatment of liver abscesses can be effected. The acute type of case, in which there is sufficient balance in the contest between the defensive mechanisms of the body and the invading organisms to enable localized abscesses to be formed, is the one which may be amenable to surgical intervention.

This surgery consists of evacuation of the pus, by a route which depends on the individual case. Gross upward enlargement of the liver indicates a posterior transthoracic approach; but should the enlargement be mainly downwards, an abdominal approach may be preferred. If by one approach pus cannot be found, the other should be tried and at the same sitting, if

possible. The liver, when exposed, may present an obvious abscess; if not, it is needled until pus is found or until its presence, in any large collection, can be excluded. When the abscess has been located, it is probably best to leave the needle in place and to make an incision beside it with scalpel or diathermy knife into the cavity, then to insert the finger to break down loculi and to arrange drainage. Patients have recovered after a simple evacuation by the aspirating needle of as much pus as possible, followed by closure of the abdomen.

Should subsidence of symptoms not occur and the leucocyte count be still raised, further exploration is indicated.

The problem of when exploration is first indicated is one shared by liver abscess with subphrenic and perinephric abscess. It is gauged by the progress of the case, with all the definable and indefinable factors which bring the surgeon to a realization that something further has to be done if the patient is to recover.

The above remarks on treatment, although directed largely towards the acute type of case, apply equally to the subacute and chronic types. These are even more susceptible to successful treatment, because toxæmia is less evident, the progress of the disease is slower, and the abscess in the liver is usually solitary.

SUMMARY OF TREATMENT.

Treatment may be briefly summarized as follows. If early diagnosis of portal pyæmia has been made (*vide supra*) and no operation has been performed on the appendiceal focus, this should be attended to forthwith and the ileo-colic vein (or superior mesenteric if necessary) ligated. Sulphonamide therapy in some adequate form should be immediately instituted. If, however, the appendix has already been removed (or an abscess drained), sulphonamide therapy alone should be tried for thirty-six hours. Should improvement not then have occurred (cessation of rigors, fall of temperature), a secondary venous ligation should be performed. At these operations any abscesses along the course of the vein should be incised and a drainage tube put down to their site.

With regard to late cases, sulphonamide should be exhibited and surgical drainage instituted when necessary and possible.

SUMMARY.

1. A case of portal pyæmia in which there were multiple hepatic abscesses that complicated acute appendicitis and in which the patient recovered after surgical drainage, is reported.

2. The literature on portal pyæmia following appendicitis is reviewed and the recorded cases of recovery are tabulated.

3. A comprehensive account of the condition, as a complication of appendicitis, is given.

4. As a result of the perusal of the literature, the following conclusions are drawn: (i) Early operative treatment of acute appendicitis would greatly diminish this already uncommon complication. (ii) The prognosis of portal pyæmia should be greatly improved by early ligation of the ileo-colic vein proximal to the clot. (iii) Liver abscess formation, while of serious import, is not necessarily fatal.

5. It is suggested that sulphonamide therapy should be of considerable service in the treatment of this disease.

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REFERENCES.

(1) G. E. Armstrong: "Introduction to a Discussion on Appendicitis", *The British Medical Journal*, Volume ii, 1897, page 945.

(2) E. A. Babler: "Pylephlebitis Complicating Appendicitis", *Annals of Surgery*, Volume lxi, 1915, page 589.

(3) R. A. Barlow: "Case of Acute Appendicitis: Suppurative Pylephlebitis: Recovery", *The Lancet*, Volume i, 1919, page 844.

(4) H. L. Barnard: "An Address on the Surgical Aspects of Subphrenic Abscess", *The British Medical Journal*, Volume i, 1908, page 371.

(5) W. A. Barnes and L. V. Pearson: "Suppurative Pylephlebitis and Hepatic Abscesses Complicating Appendicitis: A Case of Recovery", *The British Medical Journal*, Volume i, 1928, page 390.

(6) E. Beer: "Ligation of Portal Vein in Suppurative Pylephlebitis", *The American Journal of the Medical Sciences*, Volume cl, 1915, page 548.

(7) L. A. Bidwell: "Hepatic Abscess Following Acute Appendicitis", *The British Medical Journal*, Volume ii, 1910, page 507.

(8) Bitner: *Wiener klinische Wochenschrift*, 1912, page 187 (quoted by Petrén, *loco citato*).

(9) G. P. Bletchley: "Appendicitis Complicated by Hepatic Abscesses", *The British Medical Journal*, Volume i, 1908, page 1164.

(10) F. F. Boyce, R. Lampert and E. M. McFetridge: "Occlusion of Portal Vein: Experimental Study with its Clinical Application", *Journal of Laboratory and Clinical Medicine*, Volume xx, 1935, page 935.

(11) K. Braeunig: *Münchener medizinische Wochenschrift*, Volume lxviii, 1921, page 1115.

(12) H. Braun: *Beiträge zur klinischen Chirurgie*, Volume lxxxvi, 1913, page 314.

(13) G. A. Brogden: "Note on a Case of Suppurative Pylephlebitis", *The Lancet*, Volume i, 1911, page 301.

(14) L. Brown: "Suppurative Pylephlebitis", *St. Bartholomew's Hospital Reports*, Volume xxxvii, 1901, page 95.

(15) H. O. Bruggeman: "Isolated Abscess of the Liver Complicating Appendicitis", *Annals of Surgery*, Volume lxv, 1917, page 462.

(16) F. Brunner: *Korrespondenz-Blatt für schweizer Aerzte*, Volume xlviii, 1918, page 984.

(17) Brütt: *Zentralblatt für Chirurgie*, Volume xlix, 1922, page 610.

(18) J. H. Bryant: "Suppurative Pylephlebitis", *Guy's Hospital Reports*, Volume liv, 1897, page 77.

(19) A. J. Burge: "Multiple Abscess of Liver Following Appendicitis, with Report of Four Cases and Autopsies", *Iowa Medical Journal*, Volume xx, 1908, page 521.

(20) Cabot (Case 17472): "Portal Thrombosis: Pylephlebitis", *New England Journal of Medicine*, Volume ccv, 1931, page 1015.

(21) Cabot (Case 18251): "Chills and Jaundice Complicating Appendicitis", *New England Journal of Medicine*, Volume ccvi, 1932, page 1314.

(22) Cabot (Case 18472): "Abdominal Pains and Chills: Acute Appendicitis: Pylephlebitis", *New England Journal of Medicine*, Volume ccvii, 1932, page 950.

(23) F. Christopher: "Pylephlebitis of Appendical Origin Simulating Lung Abscess", *Surgical Clinics of North America*, Volume i, 1921, page 1093.

(24) Chvostek: Quoted by Gerster, *loco citato*.

(25) J. Jackson Clarke: "Two Cases of Abscess of the Liver Complicating Appendicitis", *Medical Press and Circular*, Volume i, 1911, page 198.

(26) R. Colp: "Chills in Acute Appendicitis", *Annals of Surgery*, Volume lxxxv, 1927, page 257.

(27) R. Colp: "Treatment of Pylephlebitis of Appendical Origin, with a Report of Three Cases of Ligation of the Portal Vein", *Surgery, Gynecology and Obstetrics*, Volume xliii, 1926, page 627.

(28) J. B. Deaver and J. A. H. Magoun: "Review of 5,488 Appendectomies Performed at the Lankenau Hospital of Philadelphia", *Annals of Surgery*, Volume lxxix, 1924, page 854.

(29) E. L. Eliason: "Pylephlebitis and Liver Abscess Following Appendicitis", *Surgery, Gynecology and Obstetrics*, Volume xlii, 1926, page 510.

(30) C. A. Elsberg: "Solitary Abscess of the Liver", *Annals of Surgery*, Volume xliv, 1906, page 217.

(31) A. W. Farmer: "Appendicitis Complicated by Septic Portal Thrombosis", *The Medical Journal of Australia*, Volume ii, 1923, page 8.

(32) R. H. Fitz: "Perforating Inflammation of the Vermiform Appendix", *The American Journal of the Medical Sciences*, Volume xcii, 1886, page 321.

(33) C. Franke: *Münchener medizinische Wochenschrift*, Volume cix, 1912, page 2107.

(34) A. Fromme: *Zentralblatt für Chirurgie*, Volume lii, 1925, page 450.

(35) W. George: "Appendicitis and Hepatic Abscess", *The British Medical Journal*, Volume i, 1928, page 633.

(36) A. G. Gerster: "On Septic Thrombosis of the Roots of the Portal Vein in Appendicitis", *Medical Record of New York*, Volume lxiii, 1903, page 1005.

(37) A. E. Halstead (reported by F. Christopher): "Four Cases Requiring Liver Surgery", *Surgical Clinics of North America*, Volume iii, 1923, page 973.

(38) Hempel: *Zentralblatt für Chirurgie*, Volume xiii, 1923, page 711 (quoted by Stewart-Wallace, *loco citato*).

(39) G. C. Heyd: "Changes in the Liver Associated with or Coincident with Infection of the Appendix", *The Journal of the American Medical Association*, Volume lxxxiii, 1924, page 1403.

(40) J. Hohlbaum: *Zentralblatt für Chirurgie*, Volume lvi, 1929, page 859.

(41) Holden and Moran: "Multiple Abscesses of the Liver Treated Intravenously with Mercurochrome—Complete Recovery", *Surgical Clinics of North America*, Volume iv, page 1241.

(42) Jalaguier: Case reported by Quénu and Mathieu, *loco citato*.

(43) W. D. C. Kelly: "Notes on Some Interesting Cases. Case 2—Acute Appendicitis, Liver Abscess, Recovery", *Journal of Royal Army Medical Corps*, Volume xxi, 1913, page 688.

(44) Kelly and Hurdon: "The Vermiform Appendix and its Diseases", 1905.

(45) O. Kleinschmidt: *Klinische Wochenschrift*, Volume xi, 1932, page 535.

(46) W. Körte: *Berliner klinische Wochenschrift*, 1892, page 794.

(47) H. Koster and L. P. Kasman: "Pylephlebitis", *Archives of Surgery*, Volume xxvii, 1933, page 910.

(48) F. J. Leilby: "Pylephlebitis Following Appendical Abscess", *Northwest Medicine*, Volume xxxi, 1932, page 325.

(49) M. Lepetit: *Revue de chirurgie*, Volume xlviii, 1912, page 758.

(50) H. Lilienthal: "Appendicitis with Diffuse Pylephlebitis", *Annals of Surgery*, Volume lvi, 1912, page 938.

(51) E. Loison: *Revue de chirurgie*, Volume xxi, 1900, page 522.

(52) Makrowski: Quoted by Petrén, *loco citato*.

(53) E. Melchior: *Zentralblatt für Chirurgie*, Volume liv, 1927; page 3083.

(54) Mgaloblischwli: Quoted by Walter-Sallis, *loco citato*.

(55) C. A. Morton: "Cases of Hepatic and Intestinal Surgery", *Bristol Medico-Chirurgical Journal*, 1897, page 317.

(56) A. V. Moschowitz: "Suppurative Portal Pylephlebitis", *Annals of Surgery*, Volume liii, 1911, page 549.

(57) A. V. Moschowitz and E. Moschowitz: *Archiv für klinische Chirurgie*, Volume lxxxii, 1907, page 683.

(58) J. C. Munro: "Lymphatic and Hepatic Infections Secondary to Appendicitis", *Annals of Surgery*, Volume xlii, 1905, page 692.

(59) H. Neuhof: "Experimental Ligation of the Portal Vein: Its Application to the Treatment of Suppurative Pylephlebitis", *Surgery, Gynecology and Obstetrics*, Volume xvi, 1913, page 481.

(60) A. Ochsner, I. M. Gage and E. Garside: "The Intraabdominal Post-Operative Complications of Appendicitis", *Annals of Surgery*, Volume xci, 1930, page 544.

(61) A. Ossip: "Pylephlebitis and Multiple Liver Abscesses Complicating Acute Appendicitis: Report of a Case", *Medical Journal and Record*, Volume cxxxviii, 1933, page 95.

(62) Perman: Quoted by Petrén, *loco citato*.

(63) G. Petrén: *Beiträge zur klinischen Chirurgie*, Volume xciv, 1914, page 225.

(64) E. Quénu and P. Mathieu: *Revue de chirurgie*, Volume xliv, 1911, page 519.

(65) I. Reök: *Zentralblatt für Chirurgie*, Volume liv, 1927, page 196.
(66) A. W. Mayo Robson: "A Case of Perforating Wound of the Abdomen in which the Superior Mesenteric Vein was Wounded: Ligature of Vein: Lavage of Abdomen: Transfusion: Recovery", *The British Medical Journal*, Volume ii, 1897, page 77.
(67) M. Rocher: Case presented at the French Congress of Surgery, *Revue de chirurgie*, Volume xlvi, 1912, page 759.
(68) H. Rolleston and J. W. McNee: "Diseases of Liver, Gall Bladder and Bile Ducts", Third Edition, 1929, pages 107 to 121.
(69) C. F. Schiltz: "Pylephlebitis of Appendicular Origin", *Northwest Medicine*, Volume xxix, 1930, page 36.
(70) A. Rendle Short: "Index of Prognosis and Results of Treatment by Various Authors", 1922.
(71) W. H. Snyder, M. G. Hall and A. W. Allen: "Association of Pylephlebitis and Appendicitis", *New England Journal of Medicine*, Volume ccxii, 1935, page 183.
(72) Soligoux and Roederer: Quoted by Petrén, *loco citato*.
(73) Sprengel: *Zentralblatt für Chirurgie*, Volume xxxviii, 1911, page 33.
(74) A. M. Stewart-Wallace: "Pylephlebitis Complicating Appendicitis and its Treatment by Ligature of Mesenteric Veins", *The British Journal of Surgery*, Volume xxiii, 1935, page 362.
(75) Sudeck: *Zentralblatt für Chirurgie*, Volume xliii, 1922, page 612.
(76) P. Syms: "Appendicitis, Portal Pylephlebitis and Abscess of the Liver", *Annals of Surgery*, Volume xlix, 1909, page 856.
(77) W. Thalimer: "Chills Occurring Early in Appendicitis before Operation and their Indication of Operable Stage of Pylephlebitis", *Archives of Surgery*, Volume viii, 1924, page 658.
(78) J. A. Thyne: "Abscess of Liver Secondary to Appendicitis: Operation: Recovery", *Australasian Medical Gazette*, Volume xviii, 1899, page 95.
(79) F. Treves: "A Case of Suppurative Pylephlebitis in which Laparotomy was Performed", *The Lancet*, Volume i, 1894, page 663.
(80) Verebély: Quoted by Reök, *loco citato*.
(81) J. Walter-Sallis: *Revue de chirurgie*, Volume xlix, 1914, pages 181 and 649.
(82) S. Weil: *Berliner klinische Wochenschrift*, 1920, page 270.
(83) S. West: "Meeting of the Clinical Society of London", *The Lancet*, Volume i, 1884, page 525.
(84) Wilms: *Zentralblatt für Chirurgie*, Volume xxxvi, 1909, page 1041.
(85) Wilms: *Münchener medizinische Wochenschrift*, Volume xlviii, 1901, page 1277.

INDICATIONS FOR, TECHNIQUE OF, AND RESULTS OF, TOTAL THYREOIDECTOMY.[1]

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Indications for Total Thyreoidectomy.

In general, patients under forty-five years of age are unsuitable for total thyreoidectomy except perhaps in certain cardiac conditions. The indications for the operation may be grouped under the following four headings.

A. *Thyreotoxic Conditions.*

(1) Old-standing and slowly progressing thyreotoxicosis of low grade in elderly patients.

(2) Thyreotoxicosis developing in women after the menopause.

(3) Thyreotoxicosis in men after fifty years of age.

(4) Recurrent thyreotoxicosis after previous medical treatment, operation or X-ray therapy.

(5) The masked thyreotoxicosis of middle age which is usually associated with an atrophic or impalpable thyreoid.

(6) Auricular fibrillation secondary to thyreotoxicosis.

(7) Congestive heart failure associated with or secondary to thyreotoxicosis.

B. *Non-Toxic Thyreoid Conditions.*

(1) Large colloid goitres in patients over forty-five years of age.

(2) Multiple adenomatous or cystoadenomatous goitres, especially if retrosternal.

(3) Diffuse nodular goitre in patients who are approaching middle age.

(4) Recurrent adenomata after previous operation in patients over forty years of age.

(5) The presence of myxœdema that does not respond to medical treatment.

(6) Reidel's struma causing symptoms of pressure.

(7) Conditions suspicious of Hürthle-cell tumour.

(8) The suspicion of carcinoma or sarcoma.

C. *Cardiac Conditions.*

(1) Recurrent congestive heart failure in patients who respond temporarily to rest and medication, whose basal metabolic rate is not

[1] Accepted for publication on May 12, 1938.

below −10% and who are not stout. Persistent ascites, hydrothorax, nephritis, coronary occlusion, syphilis and adiposity are definite contra-indications.

(2) Persistent auricular fibrillation not responding to medical treatment.

(3) True angina which is not controlled adequately with medical treatment.

D. *Diabetes Mellitus.*

(1) In certain rare cases of diabetes which cannot be controlled adequately with insulin and other medical treatment in patients whose basal metabolic rate is within normal limits, an increase in tolerance proportionate to the decrease in the basal metabolic rate follows on total thyreoidectomy.

(2) Whenever true diabetes is associated with thyreotoxicosis.

THE PARATHYREOIDS AND THE DEEP CERVICAL FASCIA.

Before technique is considered it is necessary to detail certain anatomical relationships of the thyreoid gland. From the physiological point of view the parathyreoids are of supreme importance and their preservation is an integral part of the technique of thyreoidectomy. An intimate knowledge of the deep cervical fascia is essential for the preservation of the parathyreoids and the safeguarding of the external and inferior laryngeal nerves. It is also of great practical importance in the operative technique.

The Parathyreoids.

For a proper understanding of the parathyreoids it is necessary to turn to embryology, since the ultimate distribution and location of these glands are intimately wrapped up in the development of both thyreoid and thymus. G. Louis Weller, junior, and Edgar H. Norris, from the Carnegie Institute of Washington, presented in 1933 and 1937 respectively the most thorough embryological investigation of the parathyreoid glands; and their researches, combined with J. R. Gilmour's 428 dissections of the thyreo-parathyreoid apparatus, present a clear picture of the anatomy of these small but important glands. The inferior parathyreoids (referred to as Parathyreoid III) are developed from the third branchial pouches in close relationship with the thymus, whilst the superior parathyreoids (Parathyreoid IV) arise from the fourth pouches in close relationship with the lateral thyreoid primordia (the old ultimo-branchial bodies) (Figure I). The descent of the thymus carries Parathyreoid III to a level at or below the lower pole of the thyreoid, whilst the fusion of the lateral thyreoid with the expansion of the central thyreoid determines the position of Parathyreoid IV to the region of the posterior border of the thyreoid at or above the level of the thyreoid isthmus.

Their anatomical positions may be briefly summarized as follows.

Parathyreoid III.—In 75% of cases Parathyreoid III (the inferior) lies free in the areolar tissue lateral to the trachea and behind the thyreoid. In 15% it is in close relationship with the thyreoid on its posterior border. In 5% it appears embedded in the lateral aspect of the thyreoid near its posterior border—a surgically dangerous position. In 5% it may lie anterior to or a little below the lower pole of the thyreoid (see Figure II).

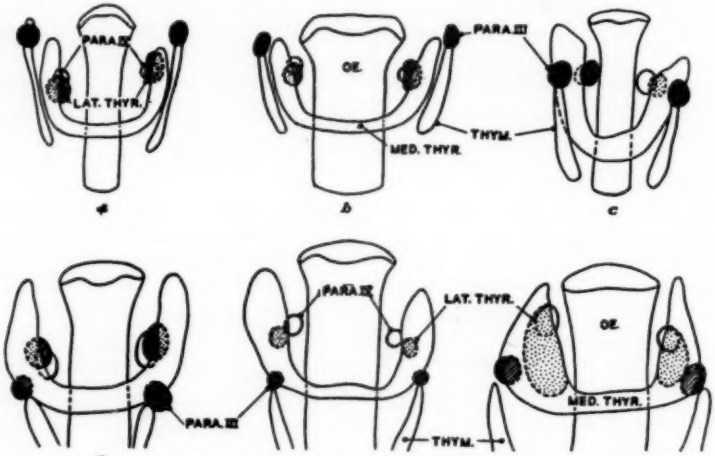

FIGURE I. An arrangement of drawings based upon selected graphic reconstructions to show the usual changing relations to the derivatives of the third and fourth branchial pouches during the branchial complex stage. All of the drawings represent anterior views. The central structure in outline represents the œsophagus; the U-shaped figure in outline, the median thyreoid; the elongated bar-like bodies in outline, the thymus. Parathyreoid III is shown in solid black (diagonals where it lies behind the thyreoid). Parathyreoid IV is in simple outline; lateral thyreoid in coarse stipple (finer stipple when it lies behind or within the median thyreoid lobe). (After Edgar H. Norris.)

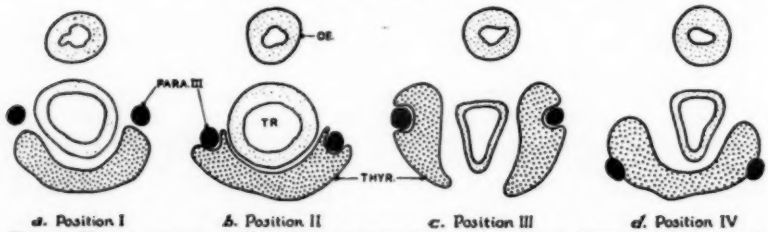

FIGURE II. A group of diagrams representing cross-sections through the thyreoid region to indicate variations in position of Parathyreoid III. These four diagrams correspond to the four position groups described in the text. Parathyreoid III is shown in solid black; definitive thyreoid is shown in coarse stipple; œsophagus, trachea (or larynx) are shown in fine stipple. (After Edgar H. Norris.)

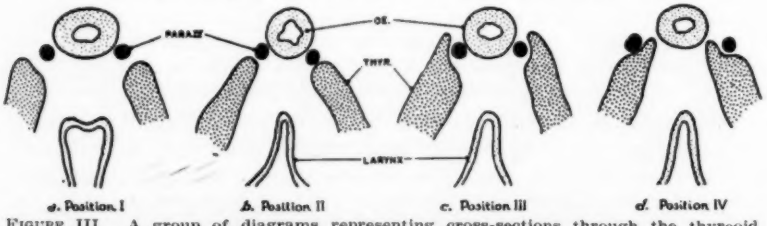

FIGURE III. A group of diagrams representing cross-sections through the thyreoid region to indicate variations in position of Parathyreoid IV. These four diagrams correspond to the four positions which are described in the text. Parathyreoid IV is shown in solid black; definitive thyreoid is shown in coarse stipple; œsophagus and larynx are shown in fine stipple. (After Edgar H. Norris.)

Parathyreoid IV.—In 85% of cases Parathyreoid IV (the superior) lies free in the areolar tissue between the posterior border of the upper pole of the thyreoid and the pharynx. In 10% it is medial to this part of the thyreoid close to the pharynx. In 5% it lies lateral to the upper pole and close to the thyreoid; this constitutes the danger position for this gland (see Figure III).

Gilmour has shown that in 87% of cases the four typical parathyreoids are present; three were present in 6% of his cases, while there was only one case in which two glands were present and two cases in which there were six glands (see Figure IV).

Parathyreoids IV are slightly smaller than Parathyreoids III, the former measuring on an average 6·2 by 3·3 by 1·4 millimetres and the latter 6·5 by 3·5 by 1·6 millimetres. It is extremely difficult to recognize them in adults, as the parenchyma is infiltrated with fat cells up to 50% of the gland bulk; this infiltration is more marked in females than in males.

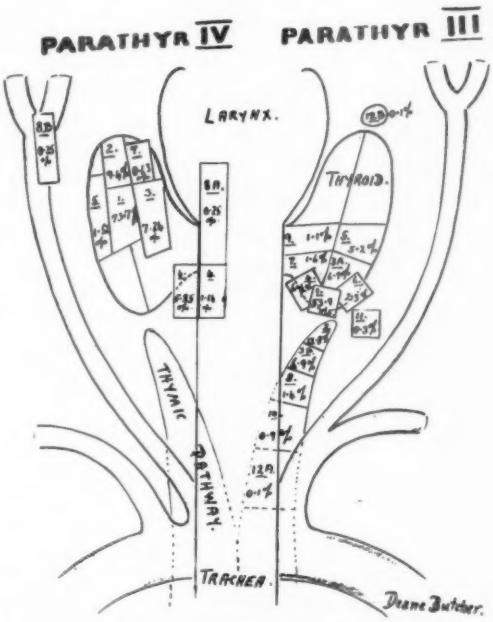

FIGURE IV. Diagram showing positions of parathyreoid glands, viewed from the posterior aspect. (After J. R. Gilmour.)

In all cases the parathyreoids are separated from thyreoid tissue by a fibrous layer. As early as in the 20 millimetre embryo mesenchyme can be seen separating parathyreoids from thyreoid. Weller points out that each parathyreoid is supplied by a separate artery, and that few, if any, connexions exist between them and the connective tissue envelope of the thyreoid gland. Weller also points out that in cases of thyreoid aplasia the parathyreoids show much more variation in number than they do in the presence of the normal thyreoid. Two or more parathyreoids arise from the splitting of one primordium and not from separate primordia. The nodules of thymic tissue, so often seen on the postero-lateral or infero-lateral aspects of the thyreoid, lie in the track of the thymus proper as it descend from the region of the third branchial cleft into the thorax. Such nodules of thymus may be mistaken for parathyreoids unless examined microscopically.

The Deep Cervical Fascia.

The textbook descriptions of the deep cervical fascia are very meagre in their details from the surgeon's viewpoint, and experience in the living has

shown the common anatomical description to be inaccurate in many ways. It was not until Meyers and Macpherson published their anatomical researches of this region in *The Medical Journal of Australia* in 1938 that a rational description was given of the deep cervical fascia and one which was in accord with the findings of the surgeon. They point out that the fundamental concept is that every muscle is covered with or enveloped in a fascial capsule, and that in the cervical region the layers of fascia correspond with the morphological muscular planes. Dealing only with the thyreoid region, we have the sterno-mastoid fascia which is carried across the mid-line of the neck (the old superficial layer of the deep cervical fascia). Next we have the sterno-thyreoid and omo-hyoid muscles which, being in the same plane, are enveloped in the sterno-omo-hyoid layer of fascia. Beneath this is the sterno-thyreoid muscle with its layer of fascia. In the mid-line these three fascial layers may fuse, depending on the amount of divarication of the infra-hyoid muscles, but generally the sterno-thyreoid fascia remains distinct (see Figure V).

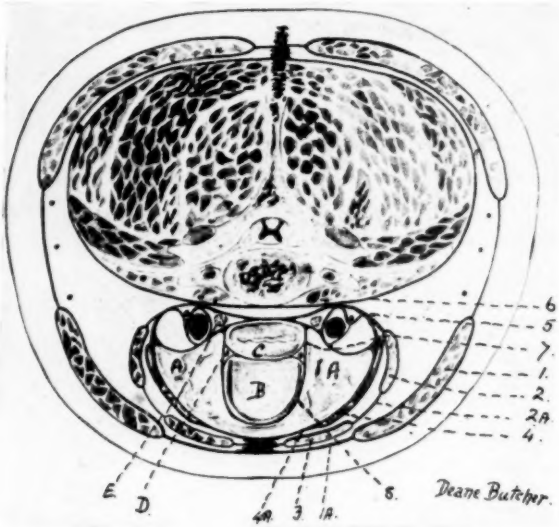

FIGURE V. Diagram of the deep fascia of the neck. (After Meyers and Macpherson.) 1 = sterno-mastoid muscle; 2 = omo-hyoid muscle; 3 = sterno-hyoid muscle; 4 = sterno-thyreoid muscle; 5 = carotid sheath; 6 = prevertebral fascia; 7 = œsophageal fascia; 8 = tracheal fascia; A = thyreoid gland; B = trachea; C = œsophagus; D = inferior laryngeal nerve; E = inferior parathyreoid gland; 1A = sterno-mastoid fascia; 2A = sterno-omo-hyoid fascia; 4A = sterno-thyreoid fascia.

The sterno-omo-hyoid fascia runs back to form the lateral part of the carotid sheath, while the sterno-thyreoid fascia forms its antero-medial part. Again, we have the inferior constrictor and œsophagus covered by a very fine fascia. The thyreoid gland is completely invested in a definite fascial covering which is formed mainly by the deep layer of the sterno-thyreoid fascia. This fascial envelope of each lobe of the thyreoid may be regarded as being pear-shaped, and the blood and lymph vessels pierce this fascia to enter or leave the thyreoid gland.

The parathyreoid, thymic remnants, external and inferior laryngeal nerves all lie outside this fascial envelope, so that if this fascia is opened along the anterior border of the sterno-thyreoid muscle, it is possible not only in theory but in practice to shell the thyreoid gland out of its bed without damage to other structures after division of the branches of the

superior and inferior thyreoid arteries and veins, also the middle thyreoid vein, as they pierce the capsule to enter or leave the gland (see Figure VI).

The only fixed point of this fascial envelope is at the region of the so-called suspensory ligament of the thyreoid which is attached to the cricoid cartilage (and at times to the trachea), so that in this region great care must be exercised.

As already pointed out, this fascial layer develops at a very early stage in the embryo and just as distinct a morphological plane exists between the thyreoid and its surrounding anatomical structures—for example, parathyreoids, inferior laryngeal nerves, inferior constrictor, œsophagus *et cetera*—as exists between the three muscular planes antero-lateral to the thyreoid gland.

It is upon the certainty of this knowledge that the detail of the technique to be described is based, and its accuracy can be judged best by the results; for since it has been adopted with minute attention to detail, no case of tetany has occurred other than in a minor degree lasting at most twenty-four to forty-eight hours, and in no case has there been any permanent damage to the inferior laryngeal nerves.

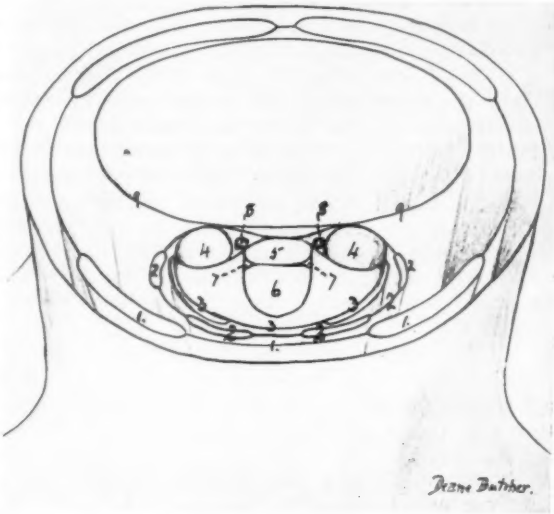

FIGURE VI. Schematic representation of the fascial layers of visceral compartment of the neck. 1 = sterno-mastoid fascia; 2 = sterno-omo-hyoid fascia; 3 = sterno-thyreoid fascia; 4 = carotid sheath; 5 = œsophageal fascia; 6 = tracheal fascia; 7 = space for inferior laryngeal nerve; 8 = inferior parathyreoid gland; 9 = prevertebral fascia.

An independent research into the fascial planes of the neck by Grodinsky and Holyoke largely confirms the anatomical observations of Meyers and Macpherson, also my own surgical experience.

The only difficulties encountered have been those cases in which there has been: (*a*) prolonged and excessive iodine medication causing an iodine thyreoiditis, (*b*) previous operation, (*c*) deep X-ray therapy or prolonged superficial X-ray therapy, (*d*) malignant infiltration, or (*e*) any acute or chronic infective process in the thyreoid gland.

TECHNIQUE OF TOTAL THYREOIDECTOMY.

Points for consideration in the technique of total thyreoidectomy are: (*a*) a minimal scar, (*b*) preservation of the parathyreoids, (*c*) safety of the inferior laryngeal nerves, (*d*) complete ablation of the gland, (*e*) minimal risk.

1. A transverse skin incision is made over the prominent thyreoid, preferably nearer the upper pole than the lower pole of the gland and overlapping the sterno-mastoid muscles by 3·0 centimetres. The fascial layers are divided down to the muscles, vessels being clamped and tied off as met with.

2. The deep fascia is dissected up and down after it has been incised along the anterior borders of the sterno-mastoid muscles. The flaps so cleared are retracted with Allis forceps placed in the mid-line close to the exposed muscles.

3. The sterno-hyoid muscles are separated in the mid-line; on the right side the muscle is retracted and the anterior border of sterno-thyreoid muscle is identified.

4. The fascia along the edge of the sterno-thyreoid is incised and with a blunt dissector introduced deep to this fascia the lateral aspect of the gland is cleared. A Kocher retractor draws the fascia and infra-hyoids laterally. It is essential to clear the deep part of the sterno-thyreoid muscle and its fascia from the gland, thus exposing the carotid sheath.

5. The upper pole of the gland, having been cleared, is pushed medially and the blunt dissector frees it from the carotid sheath. The dissector is then swung forward beneath the fascial capsule and the medial aspect of the upper pole is cleared. The blunt dissector is next passed under the superior thyreoid blood vessels, which are doubly ligated and divided after they are clamped.

6. The upper pole is then lifted forwards, with a gauze swab the fascia is pushed back from its medial, posterior and lateral aspects, and a watch is kept for the superior parathyreoid, which in 90% of cases can be pushed back with the fascial sheath. Vessels as seen are clamped close to the gland and divided.

7. The fascia still being pushed away from the gland with a gauze swab, the lower pole is elevated, and the veins are identified and clamped close to the gland. The inferior parathyreoids are deep to the fascial capsule and are seldom seen. The inferior laryngeal nerve always lies outside the fascial capsule and should seldom be seen.

8. The gland, held between left thumb and forefinger, is swung medially as the vessels are divided, and by blunt dissection close to the gland "skinning" of the trachea is prevented. The trachea is cleared across the mid-line.

9. The surgeon and first assistant change sides, all clamped vessels are tied off, a gauze swab is placed in the space so left, and the second assistant then crosses to the other side of the table, having removed his retractor.

10. The first assistant swings the gland back to the right side and makes it tense while the sterno-thyreoid fascia is incised.

11. If a pyramidal lobe is present, this is freed and ligated off.

12 to 17. Subsequent steps on the left side are then followed as described for the right side (steps 4 to 9) and the intact gland is wholly removed.

18. The gauze swab is removed from the right side and complete hæmostasis is verified. The same is done on the left side. If it is decided to use drainage, a tube is inserted through a central stab incision in the lower flap.

19. The infra-hyoid muscles and fascial capsule are coapted by a figure-of-eight stitch of catgut.

20. The Allis forceps are removed, the deep fascia including the platysma is sutured with catgut, and the skin edges are approximated with interrupted non-absorbable stitches.

21. Wound toilet is attended to and dressings are applied.

22. A lighted laryngoscope is passed, mucus is sucked out of pharynx and larynx, and the vocal cords are inspected before the patient leaves the theatre.

Advantages of the Technique Described.

It is claimed that by strict attention to this technique:

1. The minimal scar is obtained (alternate sutures are removed on the fourth day and the remainder on the fifth day).

2. There is a minimal risk of injury to the parathyreoids. In 10% of cases an upper parathyreoid may be removed (usually in cases with knobby glands), but the lower is always preserved. In no case has there been any but transient tetany cleared by an intramuscular injection of calcium gluconate, and only 2% of patients have shown any signs at all. Parathormone or "A.T. 10" has not been required.

3. The inferior laryngeal nerves are safeguarded, being seldom seen, and only transient paresis has occurred in 3% of cases; it usually clears in about one week.

4. The gland is completely ablated in a one-stage operation.

5. The whole proceedings can be completed rapidly (twenty-five to thirty minutes) and even in difficult cases the operation time should not exceed forty-five minutes.

6. Removal of mucus from the pharynx and larynx relieves the nurses of any immediate anxiety when the patient is returned to bed.

7. The risk of operation is relatively low, as no patient is refused operation, provided suitable pre-operative treatment has been carried out. In 100 cases including every risk the mortality is 4·5%. This is certainly higher than my mortality with the subtotal removal, but the results in the type of case treated are immeasurably superior to those of the subtotal operation and the extra risk is justifiable. In the worst risks two-stage operations are indicated.[1]

RESULTS.

The first 25 cases covered a period extending from October, 1936, to December, 1937, except for the first case, in which operation was performed in July, 1935. The follow-up notes are all complete to within a few weeks ago in every instance, and sufficient time has now elapsed to evaluate and verify results. Details of later cases will not be given, as they conform very closely to those now recorded in the following analysis.

In this series there was but one death, and thus the mortality rate has been only 4%. This figure was surprising in view of the fact that in nine of the cases there was grave cardiac disturbance in addition to severe thyreotoxicosis, and in eight others an established thyreotoxic condition dominated the clinical picture. Of the other cases in the first series one was carcinomatous, one a case of myxœdema, and the remaining six were cases of

[1] Subsequent experience with a much larger series of cases shows a mortality rate of 3%.

Table showing Analysis of Twenty-five Cases in which Total Thyreoidectomy was Performed.

No.	Date.	Sex.	Age.	Observations Before Operation.							Observations After Operation.				
				Goitre.	Duration.	Toxicity.	Weight.	Major Symptoms.	Pulse	Basal Metabolic Rate.	Weight.	Pulse.	Basal Metabolic Rate.	Thyreoid Substance.	General Condition.
1	31/7/35	F.	58	Small, hard.	3 years.	Severe.	st. lb. 6 4	Fibrillation for nine months under constant treatment in bed.	140	+50%	st. lb. 7 6	80 Regular.	−12%	Gr. ½ *b.i.d.*	Has resumed active life.
2	7/10/36	M.	50	Small, hard.	2 years or more.	Severe.	7 2	Has been in hospital for five months under medical treatment, including deep X-ray therapy. Put on 1 st. weight, but heart still fibrillating.	92 to 116 Very irregular	+25% +7% +7%	9 12	66 Regular.	−28% −17%	21/4/37: Myxœdema. 8/10/37: gr. ½ *b.i.d.*	Left eye enucleated for glaucoma. Back to work.
3	20/10/36	F.	51	Large cysto-adenomatous retrosternal.	Many years.	Mild.	8 1	Was in hospital nine months ago for congestive heart failure. Returned with recurrence. Has been invalid for over a year.	110 Irregular.	+12%	9 13	-86	±0	Gr. ½ *t.i.d.*	Very well and earning living again.
4	—/11/36	F.	56	Very large cysto-adenomatous.	24 years.	Mild.	15 3	Dyspnœa on exertion and irregular heart action for three years.	96 Irregular.	+5%	14 11	84 Regular.	—	Gr. ii *b.i.d.*	Finds the larger dose suits her best. Maintenance dose of tincture of digitalis, ten minims, *t.i.d.* Has not felt so well for years.
5	—/11/36	F.	40	Small right lobe. Left lobe + +.	5 months.	Mild.	6 3	Loss of weight, palpitation and nervousness.	112	+17%	6 11	80	−15%	Gr. i *b.i.d.*	Left lobe carcinomatous. Has had two courses deep X-ray therapy. Still has palpable glands three years later. Otherwise well.
6	12/12/36	F.	55	Very small and fibrotic.	10 months.	Severe.	7 2	Lost five stone. Very breathless and excitable, auricular fibrillation for four months. Is taking tincture of digitalis, 20 minims, every four hours. Has had a life of worry and several operations.	140 to 160 Irregular.	Could not do it.	10 8	80 Regular.	−12%	Gr. 1¼ *b.i.d.*	Now feeling better than for years and leading an active life. Was operated on for intestinal obstruction three months after the thyreoidectomy.

Table showing Analysis of Twenty-five Cases in which Total Thyreoidectomy was Performed.—Continued.

				Observations Before Operation.							Observations After Operation.				
				Goitre.	Duration.	Toxicity.	Weight.	Major Symptoms.	Pulse	Basal Metabolic Rate.	Weight.	Pulse.	Basal Metabolic Rate.	Thyreoid Substance.	General Condition.

Table showing Analysis of Twenty-five Cases in which Total Thyreoidectomy was Performed.—Continued.

No.	Date.	Sex.	Age.	Observations Before Operation.							Observations After Operation.				
				Goitre.	Duration.	Toxicity.	Weight.	Major Symptoms.	Pulse	Basal Metabolic Rate.	Weight.	Pulse.	Basal Metabolic Rate.	Thyreoid Substance.	General Condition.
7	12/12/36	F.	52	++ and firm.	Unknown.	Yes.	st. lb. 11 10	Palpitation for five or six years. Very breathless for eight months and heart bad. Put on one stone last year. Fibrillation present.	130 to 160 Irregular.	+33%	st. lb. 12 8	94 Regular.	−18%	Gr. ii *b.i.d.*	Occasional palpitation controlled by tincture of digitalis, 20 minims, *t.i.d.* for two or three days. Otherwise feels well, and is back at work.
8	2/3/37	F.	33	Large colloid cysts on left side.	First noticed 12 years ago.	No.	7 12	Had partial thyreoidectomy seven years ago, right lobe. Recurred on left side three years later, and growing rapidly.	80	−13%	8 3	63	−16%	Gr. ½ *b.i.d.*	General condition excellent. No sign of myxœdema.
9	9/3/37	F.	62	Large and nodular.	Many years.	Severe.	6 10	Losing weight for two years. Palpitation and nervy. Heart fibrillating.	120 Irregular.	Too ill.	—	—	—	—	Died suddenly twenty minutes after operation as a nurse was giving saline glucose *per rectum*. Condition during and after operation caused no worry.
10	6/4/37	M.	56	Moderate general enlargement.	Several years.	Severe.	8 2	Lost two stone in three years. Palpitation *et cetera* for eighteen months. In hospital for two months under medical treatment for persistent fibrillation.	80 to 120 Irregular.	+23% −1% +13% −9% +20%	10 9 10 3	48 Irregular. 70 Regular.	−22% −12%	Nil. Gr. ½ *b.i.d.*	Six weeks after operation showed early myxœdema. Condition excellent two years after operation, and has been back at work since three months after leaving hospital.
11	13/4/37	M.	34	Two very large colloid cysts.	Since six years of age.	No.	12 1	In 1925 had large swelling removed from right side. Left side and centre have increased since then.	74	−10%	12 12 12 3	76 74	−23% −14%	Nil. Gr. ii *b.i.d.*	Six weeks after operation showed definite myxœdema; now back to to normal.
12	13/4/37	F.	42	Huge adenomatous goitre.	20 years.	Slight.	6 13	Lost 2½ stone in past few years. Tires easily, nervy, attacks of palpitation.	82	+1%	8 8	76	+7%	Gr. i *b.i.d.*	Has lost all symptoms and feels better than she has for many years. Recently confined without trouble.

Tables showing Analysis of Twenty-five Cases in which Total Thyreoidectomy was Performed.—Continued.

No.	Date.	Sex.	Age.	Observations Before Operation.							Observations After Operation.				
				Goitre.	Duration.	Toxicity.	Weight.	Major Symptoms.	Pulse	Basal Metabolic Rate.	Weight.	Pulse.	Basal Metabolic Rate.	Thyreoid Substance.	General Condition.
13	27/4/38	F.	41	Large cysto-adenomatous goitre.	Many years.	Yes.	st. lb. 6 7	Had a goitre operated on six years ago. Recurred one year later. In last six months has lost half a stone in weight. Palpitation, nervy *et cetera*. Unable to work.	96	+29%	st. lb. 7 2	86	−2%	Gr. ½ *b.i.d.*	General health now first class, and leads a busy life again.
14	30/6/37	F.	49	Huge nodular goitre.	25 years.	Yes.	6 10	Has had a big goitre for years but in last nine months nervy, palpitation, tremor and loss of weight.	126	+20%	8 4	80	−9%	Gr. ¾ *b.i.d.*	Has lost all symptoms. Feels first class and resumed a very busy life.
15	30/6/37	F.	40	Large adenoma right lobe and retrosternal.	30 years.	No.	12 4	Was operated on at ages of 12 and 23 years for goitre. Swelling on right side persisted and is increasing.	84	Not done.	11 10	80	—	Gr. 1 *b.i.d.*	Feels and looks very well. No symptoms.
16	30/6/37	F.	38	Multiple adenoma; large goitre.	10 years or more.	No.	9 0	The goitre is increasing in size and very unsightly.	80	Not done.	9 11	66	—	Gr. ½ *b.i.d.*	Feels very well and leads a busy life.
17	23/6/37 7/7/37	F.	48	Very large nodular goitre.	All her life.	Severe.	10 12	For past eleven months breathless, nervy *et cetera*. Admitted in advanced condition of congestive heart failure. (Did a two stage operation.)	140 to 160 Irregular.	+48% +24%	12 12	64	—	Gr. ½ *t.i.d.*	Appeared to be *in extremis*, but settled well after four weeks' treatment. Has since returned to a busy home life.
18	12/7/37	M.	62	Very hard nodular goitre.	Many years.	Severe.	7 4	Lost lot of weight in six months. Breathlessness and tremor. Persistent fibrillation.	120 to 140 Irregular.	+69% +36%	10 10	74 Regular.	—	Gr. ½ *b.i.d.*	Has resumed his old activities and feels well.
19	13/7/37	F.	25	Large colloid goitre.	Since 12 years of age.	No.	12 2	Has been treated for three years as myxœdema, but is putting on weight and shows usual syndrome still.	80	−12%	12 12 13 10	76 80	−22% −22%	Gr. ½ *b.i.d.* Gr. 1½ *t.i.d.*	Although she has put on weight, states she feels much better and is now mentally alert.

Table showing Analysis of Twenty-five Cases in which Total Thyreoidectomy was Performed.—Continued.

Observations Before Operation.

Table showing Analysis of Twenty-five Cases in which Total Thyreoidectomy was Performed.—Continued.

No.	Date.	Sex.	Age.	Observations Before Operation.							Observations After Operation.				
				Goitre.	Duration.	Toxicity.	Weight.	Major Symptoms.	Pulse	Basal Metabolic Rate.	Weight.	Pulse.	Basal Metabolic Rate.	Thyreoid Substance.	General Condition.
							st. lb.				st. lb.				
20	13/7/37	F.	52	Large adenomatous goitre (retrosternal).	22 years.	No.	8 8	Growing very rapidly of late, especially on left side. Has a hypochromic anæmia.	80	+1%	8 7	84	−6%	Gr. ½ *b.i.d.*	General health much improved. Blood now normal.
21	7/8/37	F.	49	Very large adenomatous goitre.	20 years or more.	Yes.	9 1	Attacks of palpitation and nerves for years. Worse in last six months, and goitre is enlarging. Has become tremulous.	108	Not done.	10 9	76	—	Gr. ¼ *b.i.d.*	Two months after operation was obviously subthyreoid. Also some symptoms of parathyreoid deficiency, which cleared with Vitamin *D*. Now feels very well.
22	24/8/37	F.	65	Nodular goitre.	Many years.	Yes.	8 7	Lost one stone weight in past year; great weakness, palpitation and tremor.	116 Irregular.	Not done.	9 3	74 Regular.	—	Gr. ½ *b.i.d.*	Now feels very well and has resumed all her old activities.
23	21/9/37	F.	46	Very large cysto-adenomatous goitre.	12 years.	Yes.	10 4	In past four months losing weight. Palpitation, nervy, weakness.	96	Not done.	11 3	80	—	Gr. ½ *t.i.d.*	Husky voice for six weeks after operation. Felt very well until had severe hæmorrhage from fibroid in February, 1938, and died after operation for this.
24	28/9/37	F.	41	Huge adenomatous goitre.	28 years.	Yes.	11 3	Weakness and palpitation for one year. Nervy and tremulous.	110	Not done.	14 1	76	—	Gr. 1½ *t.i.d.*	Two months after operation showed severe myxœdema and also developed tetany. Has gradually improved under treatment.
25	15/11/37	F.	53	Large firm thyreoid with big adenoma on right side.	14 years.	Yes.	11 12	Had X-ray treatment fourteen years ago. Symptoms cleared, but not the goitre. Eight months ago had shock and has since lost weight. Nervy, palpitation and tremor. Unable to do anything.	108	+27%	12 2	90	—	Gr. ¼ *b.i.d.*	Has lost all symptoms and resumed an active life. Feels splendid.

advanced multiple adenomata with varying degrees of distortion of the trachea or with intrathoracic growth. In past years I had been tempted to perform a complete thyreoidectomy, but was held back by the disinclination to produce deliberately a surgical myxœdema. However, the fact that the condition of many patients showing evidence of subthyreoidism after almost complete removal of the gland proved to be easily controlled by small doses of thyreoid substance given by mouth, rather altered my ideas as to the control of surgical myxœdema.

It is to be clearly understood that when reference to "total" thyreoidectomy is made, the whole gland has been completely removed and no slice has been left on one or other side at the hilum or any pyramidal lobe or part of the upper pole. In every case assistants and onlookers have been quite satisfied that no remnant of gland was left behind. This point is stressed because some surgeons perform what purports to be total thyreoidectomy, but portion of the gland is retained. In none of the cases was there any damage to the recurrent laryngeal nerves, although in some huskiness of voice was present for a week or so, and in only one instance was there subsequent tetany of any noteworthy degree.

In the first case in 1935 the operation was performed at the request of a colleague physician upon a patient who had been under his care for some eight or nine months with cardiac breakdown, the result of a chronic low-grade thyreotoxicosis of some years' standing. The patient had spent six months in bed because every time an attempt was made to get her up she suffered a relapse. The operation was performed without difficulty, and three weeks later she was allowed up and left hospital after another week, taking half a grain of thyreoid extract twice a day. She has since led a useful, active life and last year enjoyed a trip to England.

The second case was that of a man of fifty years, who had been admitted to hospital with auricular fibrillation of some months' standing. He was found to have a moderate enlargement of the thyreoid and a basal metabolic rate of +25%. After two weeks' rest and medical treatment, deep X-ray therapy was commenced, as he was thought to be too bad a risk for the surgeon. He had six weeks of this treatment, but a month later was referred for a surgical opinion as the fibrillation was no better, although he had put on eight pounds in weight and his basal metabolic rate was only +7%. After a further month of medical treatment it was decided to do a partial operation, but as he reacted so well to premedication and local anæsthesia the procedure was extended to a total thyreoidectomy. He stood the operation well, and three weeks later an electrocardiogram showed that sinus rhythm had returned and myocardial function had improved. He did not report back for six months, and then only because of acute glaucoma in a blind left eye, which was then enucleated. His basal metabolic rate was found to be −28%, his pulse was regular, and its rate was 66 in the minute; he had put on nearly one stone in weight, but showed an excellent clinical picture of myxœdema. He has since kept well on half a grain of thyreoid extract twice a day.

Emboldened by the success in this case, I gave consideration to a woman, fifty-one years of age, who had been in hospital nine months previously with congestive heart failure and had again been readmitted. It was noticed she had a small goitre, but at operation we found a very large retrosternal tumour with compression of the trachea. Seven months later her basal metabolic rate was +3%, her pulse was regular, its rate was 88, and she had resumed her work, carrying on satisfactorily. Sixteen months after operation her basal metabolic rate was ±0%, and her pulse rate 86, and "she felt as well as she ever did" and had worked uninterruptedly. She kept a balance on half a grain of thyreoid substance three times a day.

The fourth and sixth patients both suffered from thyreotoxic conditions with cardiac breakdown. The fifth was a patient with thyreotoxicosis who on operation was found to have carcinoma of the lower pole of the gland on the left side. Case seven was again a chronic thyreotoxic condition with fibrillation, and number eight was the first straightforward one with non-toxic multiple adenomata.

Case nine gave a check to the successful run. The patient was sixty-two years of age and had had a goitre for years, but it gave no apparent trouble until on a visit to New Zealand some six months previously she became gravely ill with auricular fibrillation. Since then she had been practically an invalid and developed the typical appearance and clinical syndrome of a very acute thyreotoxicosis. After two weeks in hospital her condition had apparently settled and operation was performed without trouble, the patient being returned to bed in good condition with a firm regular pulse of 106. She died twenty minutes later quite suddenly as a nurse was administering the usual saline-glucose rectal injection; the condition was apparently an acute cardiac dilatation.

This case illustrates one of the difficulties met with in that she would not go to a public hospital and could not afford other than intermediate fees on a low scale for a limited period, so that no basal metabolic rate estimation was done. The patient wished for early operation, and we were misled by the apparent clinical improvement into thinking she was fit to stand it.

Greater care was exercised after this, particularly with private and intermediate patients, so that no further fatality was experienced to the end of this series, although several of the patients were very bad risks.

In a review of these twenty-five cases the first striking impression is that of the relatively slow onset of signs of myxœdema. It does not become evident for six to twelve weeks after operation and not until the basal metabolic rate falls to about or below -20%.

Alteration in facial expression is the first obvious sign; then occur dryness of the skin, thickening of the hands and feet, general sluggishness, alteration of the voice *et cetera*.

The lowest basal metabolic rate we found was the -28% already mentioned, and it seems that if you can keep the rate about -15%, the patients have no sign or symptoms of myxœdema and can lead an active life.

The next point was the very small amount of thyreoid substance (whole gland) necessary for good health. In most cases half a grain twice daily suffices, while some patients keep an even balance on one grain twice a day. In a few cases up to three grains have been necessary twice a day. In all cases before operation the necessity for taking the tablets regularly and for life has been explained, and no patient has raised any objection. In most cases the patients can by varying the dosage with half-grain tablets find the amount which suits them best, and usually a dose morning and evening is found not only sufficient, but more convenient than one after each meal.

The patient with myxœdema who was operated on had not been relieved by thyreoid medication over a long period. She was only twenty-five years old and had had a colloid goitre since she was twelve years of age. She had a basal metabolic rate of -12% to -17% over the whole period of her treatment. After operation the basal metabolic rate dropped to -22%, but she is now well with one and a half grains of thyreoid substance three times a day.

Of the 25 patients reported, four were males and 20 were females. Severe thyreotoxicosis was present in 17 cases, and in nine of these the cardiac breakdown dominated the picture.

Multiple adenomata with no obvious normal gland, but with no toxicity, accounted for six cases; and there was one patient with myxœdema and one toxic with carcinoma. The unusual occurrence of carcinoma with thyreotoxicosis is worthy of note.

Eleven of the patients were in the public ward at the Royal Prince Alfred Hospital, while 14 were either private or intermediate patients.

It has been much easier to follow up the public hospital patients, as they can be written to and asked to report, and, when necessary, tests can be carried out without expense to them. Private patients will report back only if they want to (even though no fee be charged); and when feeling well, they object to the expense of a basal metabolic rate estimation, and will not attend or are not suitable persons to have this test carried out at a public hospital.

In view of the fact that many patients with extreme conditions are now subjected to the total ablation, a mortality rate of only 3% in a later series is regarded as satisfactory.

Lambie points out there is a fall in the adenylpyrophosphoric acid content of the heart muscle coincident with a rise in energy metabolism. This substance is of fundamental importance in muscular contraction, and a fall in its amount in cases of chronic thyreotoxicosis may be an important factor in post-operative cardiac dilatation and failure.

SUMMARY.

1. Indications for total thyreoidectomy are classified under the following headings: A, thyreotoxic conditions; B, non-toxic thyreoid conditions; C, cardiac conditions; D, *diabetes mellitus.*

2. A brief review of the salient points in the embryology, anatomical location and number of the parathyreoid glands is given and their relationship to the thyreoid and fascial layers is noted.

3. The anatomy of the deep cervical fascia in the thyreoid region is reviewed in the light of recent researches and its importance is stressed from the surgical aspect.

4. Detailed steps in the technique recommended and practised by the author are given, with indications as to why this fascial enucleation of the gland is of such importance; its advantages are tabulated.

5. Results of operation, with special analysis of the first 25 cases, are discussed.

6. A bibliography of the more important and the most recent literature bearing on the points raised is appended.

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With regard to the work done since February, 1938, similar acknowledgement is to be expressed with regard to the staff of the Prince Henry Hospital. Also to Dr. Geo. Stening and Dr. Norman Wyndham, who have been associated with me in the treatment of intermediate and private patients.

I am very grateful to Dr. Deane Butcher for his help with the illustrations.

BIBLIOGRAPHY.

R. J. Millzner: "Variation in the Normal Position of the Parathyroids", *North West Medical Journal*, Volume xxix, 1930, page 423; "Normal Variations in the Human Parathyroid Glands", *Anatomical Record*, Volume xlviii, 1931, page 399.

Halstead and Evans: "The Parathyroid Glandules", *Annals of Surgery*, Volume xlvi, 1907, page 489.

G. S. Williamson and I. H. Pearce: "Anatomy (Comparative and Embryological) of Special Thyroid Lymph System", *The British Journal of Surgery*, Volume xvii, 1930, page 529.

Edgar H. Norris: "Contributions to Embryology", Carnegie Institute of Washington, Volume xxvi, 1937, Number 159, page 249.

G. Louis Weller: "The Development of the Thyroid, Parathyroid and Thymus Glands in Man", Carnegie Institute of Washington, Volume xxiv, 1933, Number 141, page 95.

M. Grodinsky and E. A. Holyoke: "Fasciæ and Fascial Spaces of Head, Neck and Adjacent Regions", *American Journal of Anatomy*, Volume lxiii, November, 1938, page 367.

A. J. Walton: "Surgical Treatment of Parathyroid Tumours", *The British Journal of Surgery*, Volume xix, October, 1931, page 285.

G. Gordon Taylor and R. S. Handley: "Unusual Case of Hyperparathyroidism. Anterior Mediastinal Parathyroid Tumour Removed by Transsternal Approach", *The British Journal of Surgery*, Volume xxv, 1937, page 6.

J. R. Gilmour: *The Journal of Pathology and Bacteriology*, Volume xlviii, Number 1, January, 1939.

W. G. MacCallum: "The Surgical Relations of the Parathyroid Glands", *The British Medical Journal*, November 10, 1906, page 1282.

E. S. Meyers and R. K. Macpherson: "Arrangements of Deep Cervical Fascia", *The Medical Journal of Australia*, November 12, 1938, page 813.

C. G. Lambie: "The Thyreotrophic Hormone and its Relation to Clinical Syndromes", *The Medical Journal of Australia*, Volume ii, 1939, Numbers 23 and 24.

Hugh R. G. Poate: "Anatomy in Surgery", *The Medical Journal of Australia*, October 7, 1939, page 534; "The Present Position of Surgery of the Thyreoid", *ibidem*, October 15, 1938, page 635.

Alan Newton: "Toxic Goitre, with Special Reference to End-Results", *The Medical Journal of Australia*, August 20, 1938, page 265.

Cecil A. Joll: "Toxic Goitre", *The British Medical Journal*, August 26, 1939, page 433.

"Transactions of the Third International Goitre Conference and the American Association for the Study of Goitre", 1938.

MECHANICAL PRINCIPLES IN THE CAUSATION AND TREATMENT OF DISEASE.[1]

LECTURE II.[2]

By Fay Maclure,
Melbourne.

Fibrous Tissue and Scars.

Scar consists mainly of fibrous tissue with a thin covering of epithelium, and this fibrous tissue, which is developed during the process of repair, is characterized by a tendency to contract.

The incisional scar of an abdominal operation is narrow, raised, rigid and red in its early days; when stretched or pressed, it becomes white and its colour slowly returns. As it ages the scar changes: it broadens and flattens and becomes soft, pliable and white. It seems that at first there is a dense network of contracted fibres which imprisons lymph and blood and offers obstruction to their outflow; and as the result of the continued pulling and movement to which the scar tissue is subjected, these fibres eventually become separated and stretched so that the appearance of the scar changes.

The scar of the collar incision for goitre is raised, œdematous and overhung on its cranial side, but never on its lower aspect; a barrier of fibrous tissue holds up the lymph which is descending by gravity until such time as the fibres open out and allow the flow to proceed.

The obliquely penetrating wound of the face produced by the flying glass of a motor car windscreen, heals and leaves an unsightly œdematous lump: the fibrous barrier beneath the undermined skin prevents the escape of lymph; time, pressure and massage loosen the network and the lump flattens.

A similar sequence of events occurs when a flap of skin and subcutaneous fat is transplanted to a scarred area of the face.

When the scar is broad and deep, as it is so often after burns, the extent of the matting and contraction proves too much for simple stretching and pressure to overcome: the mass remains thick and hard, a veritable sheath of armour plate. As the result of repeated and excessive extension, the surface cracks and heals again and again, until, as epithelium does elsewhere under the influence of chronic irritation, it changes its character and becomes epitheliomatous.

But the very features of scar, density and contraction, which indirectly determine the onset of malignancy now prove its salvation, for while offering obstruction to the outflow of blood and lymph, they likewise prevent the outflow of malignant cells. It is remarkable how seldom carcinoma of this

[1] Accepted for publication on July 11, 1939.
[2] The first lecture was published in the issue of July, 1939.

type metastasizes by blood or lymph stream; and consequently the treatment consists of mere local excision as opposed to radical operation. The incision must extend through the whole thickness of the fibrous sheath, beneath which is found a layer of fat. This layer having been reached, the plate is peeled off by easy dissection, much as one peels rind from bacon, and the gap remaining is filled by an appropriate form of skin graft.

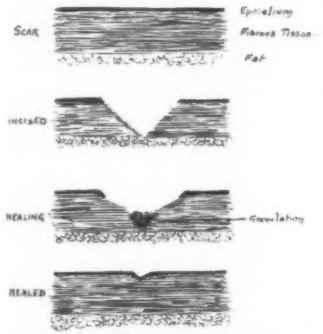


FIGURE I. Incision and healing of fibrous band.

Contraction is an inherent property of fibrous tissue, and the contraction of scar tissue and the deformities and distortions that follow in its wake are a continual source of trouble.

Often a scar has length but not much breadth, and such a more or less linear scar assumes the function of a tethering rope, limiting the range of the parts thus bound and inviting a slash across the line to free them. If an incision is made across the scar, the cleft thus produced opens out into the form of a shallow V, and the contraction is temporarily overcome, but only temporarily (Figure I). The thin covering of poorly nourished epithelium begins to advance from the edges of the wound and creep slowly. At the same moment healing is taking place in the depths, and here the development of fibrous tissue and its subsequent contraction rapidly reunite the severed fibres (Figure I).

Thus the simple section of a fibrous band left to the natural process of healing results in little improvement so far as the contraction is concerned; before epithelialization is complete fibrosis is as dense as ever and contraction just as great as before.

Epithelial Inlay or Buried Skin Graft.

It is obvious that, if reunion of the severed fibres is to be prevented, the cut ends must be epithelialized by a process more rapid than the natural one. This may be effected by an epithelial inlay or Esser's buried skin graft, in which a model of the cleft formed by severance is made in Stent's modelling compound. The model, covered by Thiersch graft with its raw surface outwards, is replaced in the cleft and buried by suturing the wound edges over it. The pressure thus generated forces the skin graft into close apposition with the cut fibrous tissue; a few days later the sutures are removed, the edges gape, the model pops out, leaving the ends of the fibres completely epithelialized and recurrence of contraction is prevented (Figure II).

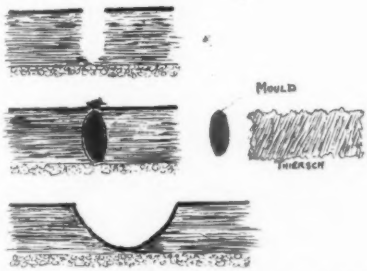


FIGURE II. Epithelial inlay. Esser's buried skin graft.

A better result is obtained by completely excising a section of the fibrous band and filling the gap either with a Wolfe graft assisted by sponge

pressure, or by means of a pedicled or tube pedicled skin flap. The severed fibres are separated by the intervening area of normal skin, and the contractile power of the two sections of fibrous tissue, which are thus prevented from joining forces, is largely dissipated in the elasticity of the healthy tissue between (Figure III).

The reverse procedure is seen in gynæcological operations designed for the repair of birth injuries. In the pelvic floor of the female are two outlets controlled by the sphincteric action of several muscles which have as their anchorage or vantage point an intermingling or fusion of muscle at the central point of the perineum. As the result of tearing, one or more of these muscles is severed, the ends retract, the gap is filled by epithelium and soft tissues, and relaxation and loss of control follow.

The basic principles of the repair of such lesions are the removal of the intervening skin and soft parts and the reunion of the contractile fibres. Unless these two points are kept in mind the operation will end in failure; the presence of elastic tissue between will nullify the efforts of the contractile elements thus separated.

FIGURE III. Excision and Wolfe graft.

When a scar has breadth as well as length, when it is extensive, as is frequently the case after burns, the fibres run in many directions, but in general they run towards a central point. If tension is applied at various points on the periphery of the scar, blanching will take place along lines, and these lines give a rough guide as to the direction of contraction in that particular sector. A study of these lines of force shows that they have a centripetal tendency; in other words, there is in a broad flat scar a circumscribed area from which contraction radiates in all directions.

Whilst the ideal remedy is the removal of the whole of the fibrous tissue, this procedure will often be impracticable or inadvisable, and in such a case the next best thing is to excise this central area and fill the gap with a whole skin graft.

Circular Scars.

Fibrous tissue is often in circular form. In the types of scar so far discussed the fibres end in normal tissues, and at worst the contraction pulls healthy parts from their natural positions and limits their range of movement.

In circular scar, however, the fibres are, in effect, endless; the result of their contraction is constriction, and the consequences of this are frequently disastrous.

The harm that is produced by contraction of circular scar is dependent on the relation of the amount of fibrous tissue to the size of the lumen involved. For example, the small degree of fibrosis developed in excision and end-to-end suture of the colon may cause little alteration in the size of the canal. Whereas in ulcerative colitis the great amount of fibrous tissue resulting from oft-repeated inflammation and ulceration may so constrict the transverse colon as to produce obstruction, a minimal amount of scar may so narrow a biliary duct as to place the sufferer's life in danger.

Fibrous tissue is the bane of the plastic surgeon. It is essential that he should have knowledge of its ways and should be able to anticipate and make allowance for the results of contraction. The circular scar, particularly

when it affects the orifices of auditory meatus, nares, mouth and orbit, forms one of his greatest problems. The ideal remedy is total excision and replacement by normal tissue, but in many cases this is out of the question, and, since simple excision is useless, he must have recourse to the principle of separation of fibres by incision and intervention of healthy and elastic skin between the cut ends.

STRICTURE OF URETHRA AND INTERNAL MEATUS.

When these ideas are applied to lesions of the urethral canal it appears that the treatment of stricture by internal or external urethrotomy can do little towards effecting a permanent cure. In actual practice, and for the reasons given above, the canal is very soon at least as narrow or narrower than before.

When the fibrotic mass is limited to an inch or less, the principle of total excision of the stricture by the Hamilton Russell operation offers the best solution of the problem (Figure IV).

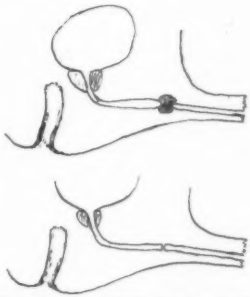


FIGURE IV. Hamilton Russell excision of stricture.

This method is inapplicable when the fibrosis extends over a greater area. The operation of choice is then the application of the principle of cutting across constricting fibres and of preventing them from reuniting by the immediate covering of their ends with epithelium. In the body of the penis this may be accomplished by "feminizing" the urethra. In this operation, the Russell artificial hypospadias operation, the fibrous ends are covered by suturing mucosa to skin on each side of the incision of an external urethrotomy (Figure V).

In the perineal portion of the urethra when the canal is at much greater depth from the surface, the writer suggests that it may be possible to adhere to this principle by adapting the buried skin graft technique to this particular problem (Figure VI).

Obstruction to the internal meatus is produced by fibrosis in the form of a bar or a circular stricture and usually in association with a small fibrous

FIGURE V. Artificial hypospadias or feminizing operation for non-excisable stricture in body of penis.

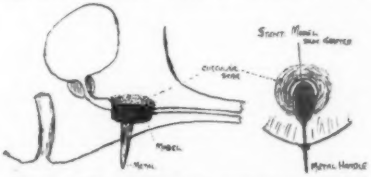


FIGURE VI. Suggested operation—buried skin graft—for non-excisable stricture in perineal urethra.

prostate. This type of obstruction is frequently dealt with by punching out a portion of tissue or by burning it out with the diathermic resectoscope. Neither of these operations is based on a sure foundation. They both involve the sacrifice of the covering mucosa, and, whilst breaking the circle of constricting fibres, do nothing to prevent their reunion. In consequence they must be put on the same plane, so far as permanency is concerned, as the operation of internal urethrotomy.

The treatment designed by Harry Harris is much more sound. It consists in the excision of a wedge of fibrous tissue and permanent breaking of the circle by the interposition of a flap of healthy mucous membrane.

The ideal procedure is total removal of the fibrous prostate by dissection under vision, but unfortunately this is possible only in selected cases.

STRICTURE OF THE BILIARY EXIT.

A calculus passing down from the kidney may become impacted at the ureteral orifice in the bladder wall, remain there for a time, slowly ulcerate, and finally escape, leaving a circular scar which by contraction will, later, cause obstruction of the duct. This sequence of events can be observed by means of the cystoscope.

Although it cannot be seen, exactly the same pathological process must take place in the biliary system—scarring and stricture of the exit of the common bile duct must often be the sequel to the passage of a gall-stone.

Disregarding infection, the two causes of jaundice associated with gall-stones are the presence of a stone in the common bile duct and a stricture of the ampulla of Vater.

For the treatment of this stricture dilatation by the passage of sounds has been advocated and presents little difficulty. The history of the treatment of an analogous condition in the urethra by similar means is a story of never-ending dilatation, and while this may be done in the case of the urethra, it cannot apply to the bile duct. The risk of recurrence after one dilatation of the common bile duct is too great to justify faith in the procedure. Since direct surgical attack on the stricture is full of difficulties and dangers, it is obvious that the safest and surest treatment is by the provision of an alternative exit for the bile. This may be accomplished by emptying the gall-bladder of stones and anastomosing it to the duodenum.

Faced with the probability of such an obstruction during operation on the biliary system, the writer has deliberately followed this course and has not had reason to regret it.

However, the diagnosis of stricture is frequently not made until, following cholecystectomy for gall-stones, recurrent jaundice, itch and fever tell the tale of biliary obstruction. A second operation discloses dilatation of the common bile duct, but palpation does not reveal the presence of a stone in the duct, and the operator is driven to the conclusion that the duodenal exit is constricted. Adhering to the principle of the two-way outlet for bile, he may elect to anastomose the common duct to the duodenum, but since the cystic duct is affected by the back-pressure, it is frequently found to be distended like a small gall-bladder, and, being more mobile than the common duct, its junction with the duodenum becomes the operation of choice.

CURLING AND ROLLING OF EPITHELIUM.

If an oblong flap of skin and subcutaneous fat is raised from the body surface and remains attached at one edge only, which constitutes its nutritive pedicle, and if this flap is kept so raised, it soon begins to curl at its edges towards the uncovered aspect (Figure VII). This process will continue until eventually epithelium meets epithelium, the bare surface is covered and the flat flap is transformed into the shape of a tube or cylinder. A similar mode of repair is seen in lesser degree on the edges of open wounds, in stripped up skin injuries, and in incisions in which the subjacent tissues have

sloughed. This rolling and curling tendency is a marked feature of epithelial repair under certain conditions—it seems that Nature abhors an uncovered area of mesothelial tissue and by means of fibrosis and its subsequent contraction rolls the edges in an attempt to cover the unepithelialized surface (Figure VII).

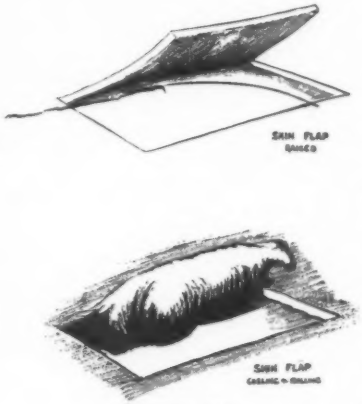


FIGURE VII.

If, however, the flap so raised has its raw surface immediately covered by a companion flap, healing follows a normal course, and no such contraction and distortion of tissues take place; the mesothelial structures having been completely covered, the fibrous tissue is not required and is developed in minimal amount.

These observations are of the greatest importance in that branch of surgery which deals with the closure of fistulæ and of communications with air sinuses with rhinoplasty and with the covering of any cavity. The skin flap to be used for these purposes may be epithelialized on its under surface prior to its transfer. If not, the principle of the companion flap should be regarded as an essential step in the operation. To achieve this object the epithelial structures in the vicinity are outlined, raised and hinged at the edges of the gap and inturned so as to line the cavity. The raw surface so presented is covered then by the transplanted skin flap, and, mesothelial tissues now being covered everywhere by epithelium, no distortion follows. To leave an area bare of epithelium on the under surface of a flap which is designed to roof over a cavity, is to invite disaster from the development of fibrosis and curling.

The same mode of repair is seen in connexion with mucous membrane. After colostomy, when the exteriorized loop of intestine is removed, the gut projects through the skin incision for a distance of a quarter to half an inch, and the cut edge of the mucosa is separated from the skin by the peritoneal coat of the colon. Now neither skin nor mucous membrane grows over this space, but the mucosa curls and the intervening mesothelial tissues fibrose and contract, and gradually approximate the mucosa and skin until epithelial tissues meet (Figure VIII).

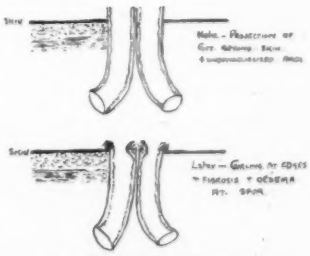


FIGURE VIII. Colostomy.

The spur or dividing wall between the two intestinal tubes consists of a mucous layer of each channel separated by the several coats of the intestine and the tissues of the mesentery. These mesothelial tissues constitute a bare area in which circulatory stasis occurs according to the principles enunciated in the first lecture. As a result, they become œdematous and rigid, and for that reason often present difficulties in the application of a clamp for the crushing

of the spur. Eventually fibrosis and contraction approximate the two edges of mucous membrane until these fuse and cover the bare area (Figure VIII).

In this process of healing by second intention much fibrous tissue is laid down, and in consequence circular scar surrounds each lumen and contracts so that the canal which originally admitted four fingers is reduced to the size of one.

ANASTOMOTIC ULCER.

Several points of importance in the technique and sequelæ of anastomosis of mucous lined surfaces arise from these observations.

Primary union of mucous membrane reduces the amount of fibrous tissue and consequently the degree of stricture is minimal.

Union by second intention is dependent on the contraction of fibrous tissue; and if the approximation of the edges of the mucosa is not brought about quickly, the intervening bare area of mesothelial tissues becomes the site of stasis of blood and lymph, leading to cessation of repair, with the consequent development of anastomotic ulcer.

In anastomosing jejunum to stomach it is easy to secure perfect apposition of mucosal edges on the posterior aspect of the junction—it is much more difficult to make sure of invagination of mucous membrane and to prevent evagination of jejunal lining along the anterior suture line.

In support of these contentions it is the writer's experience that gastro-jejunal ulcer has occurred on this aspect of the anastomosis, and that the ulcer presents the characters of œdema, rigidity, pallor and indolence, which have been shown to mark stasis elsewhere.

DUCT ANASTOMOSIS.

Anastomosis of ducts with different organs is necessary on occasions. As examples may be instanced the transplantation of ureter in partial cystectomy, the anastomosis of ureter with rectal mucosa for *ectopia vesicæ* and for other conditions, the making of a urethral meatus in amputation of the body of the penis or of a perineal opening in total removal of the organ, the junction of the cystic duct with the duodenum, and in operations upon salivary fistulæ.

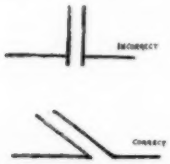


FIGURE IX. Duct anastomosis.

These ducts all possess a very small lumen, and the circular fibrosis which accompanies their anastomosis, if anything more than a minimum, will constrict the new ostium, and, should this occur, obstruction to the outflow will exert a back-pressure on the secreting organ and lead to disastrous complications. Many otherwise perfect operations have been ruined by failure to envisage the effect of circular scar and stricture.

One point in technique emerges from the discussion on the healing of a colostomy, namely, in order to minimize the formation of fibrous tissue, there should be immediate and complete apposition of the epithelial edges by careful and accurate suture (Figure IX).

Another point concerns the direction of section of the duct. If cut across transversely, the open end of the duct presents the smallest possible circumference. Any contraction of this circumference is in effect a stricture. Therefore, in order to counteract the effect of even the minimum of contraction at the junction, section of the duct should always be made in the long oblique direction (Figure IX).

DYNAMIC OBSTRUCTION.

In the second stage of the Mikulicz-Paull operation, when the clamp is applied to the spur (Figure X) in order to crush or break down the dividing wall between the two colonic canals, a set of symptoms may arise which are very disturbing, namely, abdominal colic, vomiting, and stoppage of discharge from an intestine which previously was acting regularly. These are the symptoms of obstruction, and are often so severe as to make the surgeon fear that he may inadvertently have included in the clamp a wandering loop of small intestine. These symptoms usually subside in two or three days and all ends well.

Somewhat similar events occur in the abdomino-perineal resection when the abdominal anus may refuse to act for three or four days (Figure X). In transverse colostomy, whilst a passive discharge may occur on immediate opening of the bowel, active discharge from the colon does not occur for some days (Figure X). In Richter's hernia where only one-eighth of the circumference of the bowel may be involved, the symptoms often resemble those which occur when the whole lumen is obstructed (Figure X).

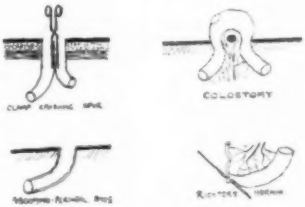


FIGURE X.

From these observations it appears that, quite apart from infective causes, simple mechanical trauma may set up a condition of dynamic obstruction of the alimentary tract above the lesion.

These facts should be borne in mind when incision, excision or anastomosis has been performed or where obstruction has existed. Coercive methods, such as the use of free fluids, early aperients and of hypodermic intestinal stimulants, are wrong and only add to the trouble. Any measures designed for the decompression of the tract above the lesion, whether by duodenal tube or by enterostomy or caecostomy, should not cease with the operation, but should be maintained until normal peristalsis is restored.

DUODENAL STUMP LEAK.

Duodenal leak is still the bugbear of gastrectomy, and, since most capable surgeons acknowledge its occurrence, it is necessary to seek other causes than errors of technique. Into this blind-ended section of the duodenum is poured the fluid secretion from liver and pancreas. In a short space of time the amount of secretion is much greater than the capacity of this part of the tract. Should dynamic obstruction occur at the gastric junction, as the result of interference with the neuro-muscular mechanism of the jejunum, then a pressure will develop within the newly made back-water, sufficient to endanger the water-tightness of a perfectly sutured duodenal stump.

As the Rehfuss tube has proved to be the salvation of many a short-circuit operation, may not the application of the same principle of decompression prove to be the means of preventing duodenal leak by the fixation in this section of an indwelling duodenal tube entering through the anastomosis?

GASTRIC ULCER.

The appearance of a chronic adherent gastric ulcer brings forcibly to mind its similarity with the varicose ulcer of the leg: the same steep walls,

the pale grey base, the large veins above on the lesser curvature, the surrounding rigidity and œdema and the same indolent appearance. Probably behind each there lies the same mechanical cause which was discussed in the first lecture, namely, stasis of venous blood and lymph, and all the alkaline powder in the world will not cure either ulcer. Even the treatment suggests this causation, for are not many such ulcers cured by cauterization and suture? And this method does not affect the biochemistry of the tract; it is merely a means adopted for the cure of œdematous granulation tissue elsewhere, a utilization of mechanical principles for the restoration of circulation.

Operations which are designed to attack the ulcer only indirectly by altering the reaction of the gastric juices, namely, gastro-enterostomy and pyloroplasty, have few advocates today; it appears that this type of treatment has little hope of effecting a cure.

As this plan brings about drainage of the stomach, at the same time it seems that methods based on this principle alone will also fail.

The great majority of operations in favour today aim at destruction of the ulcer and not at causing it to heal. This is done either by excision or by removal of the ulcer-bearing area or even greater portions of the stomach. That is to say, those who recommend this plan of action tacitly admit that an adherent ulcer cannot be healed.

When the almost numberless and widely divergent types of operation devised for the treatment of ulcer are analysed, it is evident that one factor is common to most of them—ablation of the pylorus as a sphincter. Whether this device acts by lowering the degree of acidity or by preventing distension of the stomach is not yet proven, but it is well recognized that pyloric spasm and obstruction play a large part in disease of the alimentary tract.

During the earliest days of life, gastric obstruction in the form of congenital pyloric stenosis is always dangerous and frequently fatal, and this obstruction is considered to be due to hypertrophy of the circular muscle and to pyloric spasm. In gastric dilatation which supervenes on chronic duodenal ulcer, the obstruction is caused not so much by the stenosing effect of the fibrotic ulcer as by the superadded pyloric spasm. Excision of gastric ulcer alone frequently fails; hence this operation is usually combined with gastro-enterostomy as a means of preventing the distension and consequent damage to the resection suture which arises as the result of pylorospasm.

In this connexion it seems extraordinary that the exceedingly simple mechanical principle, the Ramstedt incision of pyloric muscle, which has proved to be such a life-saving measure in the child, has not been utilized for analogous conditions in the adult.

In duodenal ulcer such a course would not relieve the condition because the cure is achieved by the diversion of the acid stream and the cessation of the mechanical irritation of the passage of food which results from the short-circuiting operation.

ANTERIOR AND POSTERIOR GASTRO-ENTEROSTOMY.

Surgeons who advocate the operation of posterior gastro-enterostomy state as one reason for their choice that drainage of the stomach is more efficient when the anastomosis is made posteriorly.

If the alimentary tract is to be regarded as a downpipe through which the contents must fall, then the position and aspect of the stoma demand consideration. But if, rightly, it is viewed as an active and contractile viscus which forces the food onwards through an opening, then it matters little whether the junction be effected on the anterior or the posterior wall of the stomach. It should be remembered also in this connexion that the position of the stoma in space is continually altering day and night with the varying attitudes of the body.

Anterior gastro-enterostomy is very much easier of execution than the posterior operation—access is better and the operation may often be done outside the abdomen—and when gastro-jejunal ulcer occurs, as come it must to every operator, any repair which may be necessary is very much more easily dealt with when the stoma lies in the anterior compartment of the abdominal cavity.

BOWEL WASH-OUT.

Abdominal distension associated with failure to pass fæces or flatus follows many operations. Whether due to adhesions, paresis, local peritonitis, ileus or any other cause, it is a distressing complication and a source of anxiety until it is overcome.

Of the simpler remedies for the relief of this condition none is better than the bowel wash-out. If left to a nurse it consists of a series of rapid fillings and emptyings, usually without the desired result. It must be remembered that the intestine has been distended for some time and is weary—its peristalsis is weak and slow, and therefore it is to be wheedled, not whipped.

The correct procedure has a rhythm and a tempo of its own. The slow mechanical dilatation of the colon by the injected fluid initiates a peristaltic wave in the distended gut, slow emptying encourages it further, and a pause of five minutes after emptying enables the wave to travel slowly along the paresed intestine. To hurry any stage usually causes a reflex inhibition of the peristaltic response and a failure of the treatment.

A bowel wash-out may be a life-saving measure, and it is the duty of the medical attendant to carry it out himself and to be prepared to spend an hour in so doing.

OMENTUM.

A traumatic or inflammatory focus within the abdomen is walled off by the adhesion of omentum or of intestine. If by the latter, all manner of complications may ensue—kinking, distension, paresis, obstruction, perforation *et cetera*—so that both the immediate effects and the sequelæ are much more serious than if the omentum is concerned. It is obviously of great advantage to have any necessary sealing and adhesion done by the omentum.

Unfortunately, the length of the omentum varies. It may be relatively short, and in association with this condition may be found a mesocolon which is not of normal length; consequently the availability of the omentum as a surgical aid may be considerably lessened by these shortcomings. For example, it may be apparent during the operation on an inflamed appendix lying in the floor of the pelvis that the omentum cannot reach it and that the inflammatory focus or the subsequent pelvic abscess will have to be shut off by coils of gut. The prognosis is considerably worse in such a case.

Again, the omentum frequently becomes bound to the site of a previous lesion or of an earlier abdominal operation. Should it be necessary to open

such an abdomen for another purpose, it is a wise precaution to unfetter such a bound omentum so that this abdominal fire brigade may be free to hasten to the later and more distant conflagration within the peritoneum. An anchored omentum is a surgical ally lost.

RENAL LESIONS.

The kidney of the ox presents on its outer surface a series of lobules separated by clefts. When it is dissected, each lobule shows cortex and medulla and a urinary tube which joins with those of neighbouring lobules and eventually forms a ureter. So it appears that the organ is a collection of miniature kidneys (Figure XI).

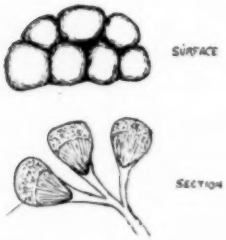


FIGURE XI. Ox kidney.

Bearing in mind the arrangement in the ox, the human kidney may be visualized as an agglomeration of renal units, and this conception of its anatomical structure leads to a clearer understanding of the treatment of certain of its lesions. For example, calculus occurring in the substance of the kidney destroys only the particular renal unit involved, and therefore in the surgeon's judgement its removal may not be imperative (Figure XII). But when the stone is in the pelvis, the obstruction to the outflow which it causes affects the whole of the kidney, and consequently its treatment and the need for its early removal as a means of preventing further damage to the kidney become a matter of the greatest importance (Figure XII).

The "renal unit" idea will also be helpful in the consideration of the conservative treatment of tumours of the upper and lower pole, of lesions of the horseshoe kidney, of double ureters, and of infections such as cortical abscess and of carbuncle of the kidney.

FIGURE XII. Renal and pelvic calculus.

In one process of estimating the renal function, indigo-carmine is injected into a vein, is carried around in the blood stream, reaches the kidney, and within a few minutes from the time of injection is excreted in the urine. The kidney apparently has the power to detect substances which are foreign to the blood stream and to excrete them selectively by way of the urine.

It may be presumed that this power extends to other foreign bodies such as microorganisms, and that one of the normal functions of the kidney is to rid the blood stream of wandering bacilli, and that bacilluria is a manifestation of this function. So long as the renal tissue is healthy, no harm can result from this process; but if for any reason the resistance of the kidney should be lowered, then the microorganisms become invaders and inflammation of the organ follows.

It is obvious, therefore, that the problem of recurrent pyelitis must be considered from two different aspects. The one involves a search for the source of the blood-borne organisms which have caused the hæmatogenous infection, this source being often an unsuspected cholecystitis, and the other requires investigation to disclose what has interfered with the mechanics of urinary outflow and thus lowered the renal resistance, whether stone, uretero-pelvic stricture, pelvic obstruction by kinking, valve formation *et cetera*.

Surgical Technique.

OPERATION FOR HARE-LIP.[1]

By Sir Robert Wade,
Sydney.

Unilateral Hare-Lip.

The operation for unilateral hare-lip is shown in the accompanying illustrations.

Figure I shows two round needles fixed into a disk handle, as figured by Veau. These two needles are set in parallel two millimetres apart.

Figure II shows the first steps of the operation. The stippled area shows where free undercutting of the *ala nasi* and of the cheek over the superior maxilla is carried out.

Figure I.

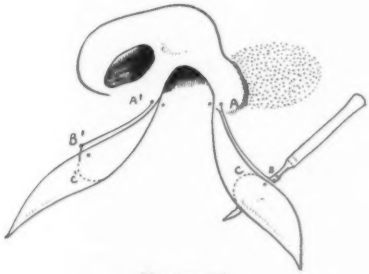


Figure II.

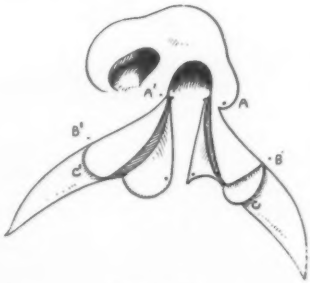


Figure III.

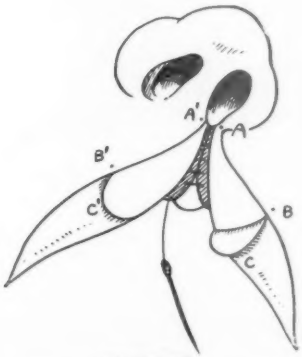


Figure IV.

[1] Accepted for publication on November 28, 1938.

The needles are inserted after due measurement at the top and the bottom of the line of intended incisions of the cleft and straddle the muco-cutaneous junction; they are used both to steady the lip while the incisions are being made and to leave a puncture mark as a site for the insertion of stitches.

A tenotomy knife is inserted at the puncture site (B) to transfix the lip, and a crescentic flap (C) is made by cutting upwards and outwards. The skin is incised from A to B and from A′ to B′ and a flap is dissected backwards, almost reaching the buccal lip mucosa. This ensures that no mucous membrane is included in the scar as a result of erratic line of incision.

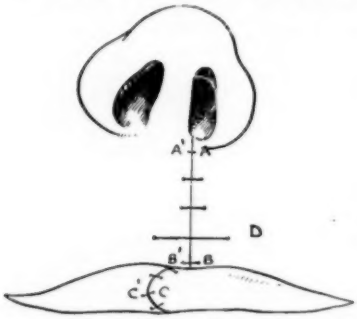


FIGURE V.

On the opposite side a similar flap of mucosa (C′) is dissected off, onto which C will later be applied.

Figure III shows the condition on completion of the steps already described.

The superfluous and irregular parts of the mucosa flaps are now trimmed off (Figure IV), enough being left to form a pad at the back of the lip to render it thicker. This mucous flap is stitched together with number 000 chromicized catgut.

As shown in Figure V, sutures of coaptation (dermal sutures) are inserted at puncture points A, A′ and B, B′, and a deep suture of relaxation (fine silkworm or dermal) is inserted through the thickness of the lip. If wished, this may be subcutaneous or may pass from the mucous membrane side (number 1 catgut). Flap C is sutured to area C′ with number 000 chromicized catgut.

Double Hare-Lip.

L. Ombredanne, writing in "*Précis clinique et operatoire de chirurgie infantile*", pages 202 to 203, describes an operation that is very satisfactory for double hare-lip.

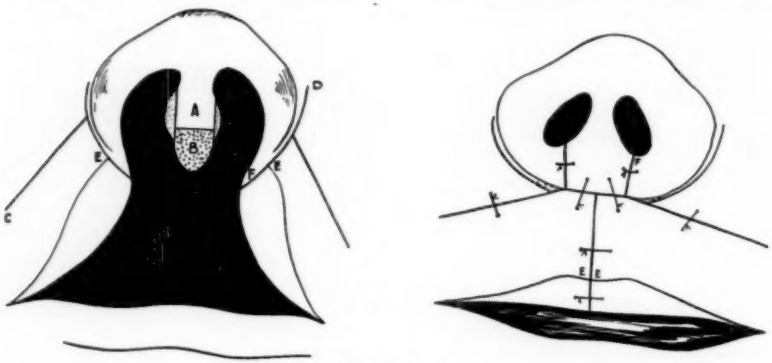


FIGURE VI.

The premaxilla is pushed back after a V-shaped resection of the vomer and secured with wire sutures. The prolabium is squared off. The lateral flaps and *alæ nasi* are freed off the maxilla by dissection. Incisions are made parallel to the muco-cutaneous junction on each flap.

The edges of the *alæ nasi* are sutured to the lateral edges of the prolabium. The flaps are brought across below the prolabium and sutured onto the lower surface and to one another.

Case Reports.

DISLOCATION OF THE CERVICAL SPINE, NECESSITATING FIXATION AT OPEN OPERATION.[1]

By James P. Ainslie,
Perth.

Extreme hyperflexion of the cervical spine not uncommonly results in fracture or dislocation of the middle or lower cervical vertebræ. Complete subluxation of the articular facets of the upper vertebra forwards on those of the lower vertebra may

Figure I. Condition on May 19, 1938. The body of the sixth cervical vertebra is dislocated forwards on the body of the seventh. The articular facets of the sixth are displaced forwards and lie in a plane anterior to those of the seventh. The body of the seventh has suffered a slight compression fracture.

Figure II. Position of the vertebræ after strong traction under general anæsthesia. The articular facets of the sixth vertebra have not been completely disengaged from those of the seventh.

occur. If this happens, reduction can be effected only by strong traction to disengage the interlocking articular facets. Once they have been disengaged by strong traction, reduction is achieved by hyperextension and manipulation of the neck, and the position is maintained by fixation in a plaster of Paris support.

[1] Accepted for publication on December 20, 1938.

FIGURE III. Condition on June 20, 1398. Forward subluxation of the sixth cervical vertebra has again occurred.

FIGURE IV. Condition on July 24, 1938. Reduction is complete and is maintained by mechanical fixation. The articular facets of the sixth now lie posterior of those of the seventh cervical vertebra.

FIGURE V. Reproduction of the condition at termination of operation showing secured fixation by stainless steel clamps over the laminæ of the sixth and seventh cervical vertebræ.

The treatment of a patient is described in whom reduction could not be achieved by these methods, and open operation and mechanical fixation had to be resorted to.

W.H., a male, aged thirty-one years, was involved in a motor cycle accident on May 3, 1938. He was admitted to a country hospital suffering from lacerations to the scalp and cerebral contusion. Sixteen days later he was sent to Perth and examination revealed deformity of the spine in the lower cervical region. Radiographic examination revealed that the body of the sixth cervical vertebra was displaced forwards on the body of the seventh and the articular facets of the sixth were lying anterior to those of the seventh (Figure I). Neurological examination did not reveal any signs of pressure on the spinal cord, and the only symptoms were limitation of movement of the neck and pain and tingling in the area of distribution of the seventh cervical nerves.

Reduction of the dislocation was attempted by the application of strong traction by a block and tackle attached to a Glisson sling after infiltration of the affected area with a 1% solution of "Procaine".

As this was not successful, a further attempt was made under general anæsthesia. Although a constant traction of seventy pounds was maintained for 45 minutes, the articular facets could not be completely disengaged; and a skiagram (Figure II) shows that although the neck had been considerably elongated, the articular facets of the sixth cervical vertebra were still in a plane slightly anterior to those of the seventh.

The site of dislocation was then exposed at operation, and each articular facet was cleaned of muscle and fibrous tissue and carefully levered into position. Hyperextension of the neck did not, however, result in the facets of the sixth cervical vertebra sliding down to the full depth of the facets of the seventh. Plaster of Paris was applied to fix the head in a position of hyperextension. A skiagram taken three weeks later showed that the dislocation had recurred (Figure III). Operation was again resorted to one June 27, 1938. Reduction was again effected, and the laminæ were brought together by gradually tightening two flanged plates of stainless steel by interlocking screws. The two vertebræ were securely fixed in a correct position, and after one plate had been applied on each side forcible flexion of the head caused no displacement of the vertebræ. Figure IV shows the position of the vertebræ after operation.

Summary.

1. The treatment of a patient suffering from dislocation of the cervical spine is described in whom the usual non-operative treatment failed to achieve reduction.

2. At open operation full reduction could not be attained and dislocation recurred in spite of fixation in plaster of Paris.

3. For mechanical fixation of two vertebræ a device (Figure V) is described which proved extremely successful in this case.

Acknowledgement.

I desire to acknowledge my appreciation to Mr. F. Gill and Mr. F. Clark for their assistance in devising a means of securing this fixation.

Post-Scriptum.

Examination in October, 1939, revealed no disability, and radiological examination showed that the vertebræ have remained in a normal position.

DOUBLE URETEROCELE.[1]

By M. S. S. Earlam,
Sydney.

Ureterocele, which is a cyst-like dilatation of the lower end of the ureter, is not a very common condition; while not necessarily causing symptoms referable to it, now and again it will be found to be the lesion responsible for the patient's symptoms, and on other occasions it will be found in patients undergoing urological investigation on account of the presence of other and more major urinary tract lesions. Most ureteroceles are unilateral and a minority bilateral; but a double ureterocele is of such extreme rarity as to warrant the publication of the present report.

Double ureterocele, that is, a ureterocele due to dilatation of the lower ends of both elements of a double ureter, must occasionally occur; but a consideration of available statistics will prove that it must be extremely rare. The frequency of duplication of the ureter, complete or incomplete, has been estimated from a study of autopsy findings at 3% by Bostroem[1] and Wagner,[2] and at 4% by Poirier.[3] In a series of 382 cases collected by Harpster, Brown and Delcher[4] complete duplication of the ureter, with two ureteric orifices in the bladder, occurred in 58·1%. On first impressions, then, the frequency of double as opposed to single ureterocele would be assessed at 58% of 3%, that is, at about 1·6%. The same three writers, however, state that of their 221 cases of complete ureteral reduplication 82% were unilateral and 18% bilateral; that is, of the 200 ureters in a hundred such patients, 118 would be double, the remainder single. In other words, $^{118}/_{200}$ of 1·6% of all ureters would be completely reduplicated, or 0·94%, and one would accordingly expect a maximum of about 1% of all ureteroceles encountered to be double. The proportion, however, will actually be smaller than this, as only one of the two orifices may undergo the changes associated with the development of ureterocele; such a case has in fact been reported by Young.[5]

A search through the available literature has disclosed no report of a case of double ureterocele, though Hinman[6] states that it may occur. Ainsworth-Davis,[7] in 1932, reported a case of ureterocele with double orifice, which he described as unique. Cystoscopy fourteen days after fulguration of the ureterocele showed one ureteral orifice only to be present. White[8] reported a similar case in 1936. In this case intravenous pyelography showed no evidence of duplication of the ureter, and White carried out suprapubic excision of a ureterocele about the size of a grape.

Clinical History.

The following is the history of the present case.

The patient, a *nullipara*, aged forty-nine years, complained that ten months previously, for the first time, "something had come down the front passage". Following this it came down with increasing frequency, until it finally descended at almost every act of urination. To get it back she had to manipulate it digitally, and this process had sometimes taken her as long as two hours. Occasionally the prolapsed material bled, and its descent was frequently accompanied by a dragging sensation in the right iliac fossa. She had mild frequency of micturition during the day, but none at night, and her condition never troubled her when she was lying down. Her medical attendant, whom she had not informed of the fact that the prolapse was associated with micturition, examined her on several occasions, but found nothing abnormal. He then advised her to return when the condition was present, found a reddish-coloured mass of tissue protruding through the external urinary meatus and resembling bladder mucosa, and referred her for investigation. In addition to the foregoing complaint she had also had for the preceding eighteen months a mild chronic urinary infection, due to *Bacillus coli*

[1] Accepted for publication on December 8, 1938.

On examination, the external urinary meatus was markedly patulous and mild pyuria was present. There were no other findings of importance.

Cystoscopy under local anæsthesia showed a large tumour with a broad base on the right side of the trigone, an undoubted ureterocele. Most of it was covered with normal bladder mucosa, but its summit was covered with bullous œdema, and in one place was ulcerated. The left ureteral orifice was normal, but the right could not be seen.

FIGURE I. Skiagram of bladder region, after injection of "Uroselectan B", showing the two large filling defects.

X-ray examination of the urinary tract revealed no evidence of calculus. Intravenous pyelography showed no abnormality of the left kidney; the right was not well visualized, but there was no hydronephrosis. Films taken of the bladder region after injection of "Uroselectan B" showed not one filling defect, as had been expected, but two large filling defects, partially superimposed, one circular, the other more ovoid, towards the right of the mid-line (see Figure I). A closer examination of the kidney and ureteric areas on the films showed appearances which were consistent with the presence of two pelves on the right side. The ureters were visualized hardly at all, but in one film portions of two right ureters, filled with dye over a length of about 1·25 centimetres (half an inch), could be seen lying side by side just below the sacro-iliac synchondrosis (these do not appear in Figure I). The shadows were too faint for reproduction, and in the absence of the two filling defects in the bladder area would probably have passed unnoticed. A diagnosis of double ureterocele was made.

Cystoscopy was carried out again the following day, after the intravenous injection of indigo-carmine. Although the dye appeared in good concentration from the left ureteric orifice, none appeared from the right side over a period of fifteen minutes after injection, and no right ureteric orifices could be seen. The closest examination showed that there was only one intravesical tumour. Only at this stage was it realized that with a large double ureterocele there would not necessarily be two tumours, the conditions being as represented diagrammatically in Figure II.

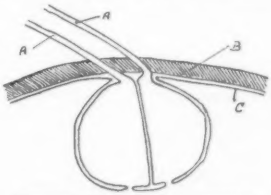


FIGURE II. Diagrammatic representation of double ureterocele. AA = ureters, B = bladder musculature, C = bladder mucosa.

In spite of the absence of indigo-carmine excretion, the facts that the right renal pelvis cast a faint shadow after the injection of "Uroselectan B" and that there was no hydronephrosis indicated conservative treatment. Accordingly, under low spinal anæsthesia, the ureterocele was removed piecemeal with the McCarthy electrotome without any particular difficulty. The tissue removed is seen in Figure III, and Dr. E. Marjory Little reported on it as follows:

"The sections show fibromuscular tissue in which are some foci of lymphoid tissue and many blood vessels. Some of the blood vessels have delicate walls, but others are well formed. The fragments of tissue are covered by transitional epithelium, which in one or two places also lines crypt-like depressions. Throughout the sections inflammatory cells (polymorphs and plasma cells) are to be seen and these are particularly numerous beneath the epithelial covering. The histological appearances are consistent with the clinical diagnosis of ureterocele."

The patient had some pyrexia during the first few days after operation and some hæmaturia, which quickly cleared up, on the tenth. Cystoscopy on the fourteenth day showed some indication of the walls of the ureterocele, in the shape of two low ridges of tissue which met at an angle at the right hand extremity of the trigone. Inside this angle were two ureteric orifices, through which both ureters were easily catheterized to their full extent. The patient left hospital the same day.

When last seen, seven months after operation, she still had mild pyuria, and complained of occasional brief attacks of "feverishness", but no localizing symptoms. Otherwise she was well.

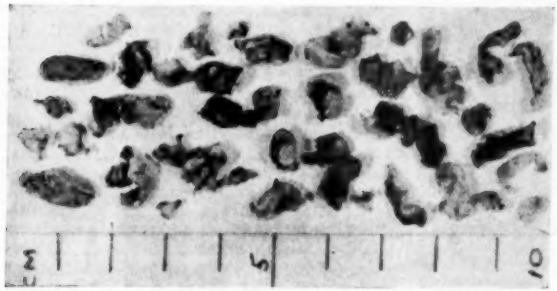


FIGURE III. Tissue removed at operation with the McCarthy electrotome.

Comment.

Before the development of the cutting diathermy current, most ureteroceles were treated by fulguration with satisfactory results. With a ureterocele of any considerable size, however, complete confidence was not always felt that the outcome, following provision of better drainage by a linear fulguration extending upwards and laterally from the ureteric orifice, would be completely satisfactory, though usually it appeared to be, and several surgeons have felt that in some of these cases complete removal of the ureterocele by suprapubic operation was preferable. Foley,[9] in 1931, devised a wire snare, by passing a current through which, after its passage through the operating cystoscope, he was able to amputate ureteroceles of moderate size. This procedure was a very definite advance on either suprapubic excision or cystoscopic fulguration. The subsequent development of the McCarthy electrotome places in our hands an equally efficient means of transurethral treatment. By both methods a ureterocele of any size can be cleanly and completely removed under full visual control, though Foley's snare might be technically more difficult to use in the treatment of unusually large tumours.

Large size is no indication for a suprapubic attack. The ureterocele in the present case was probably as large as would ever be encountered, but its removal was not associated with any particular difficulty, so that, except in the presence of associated pathological conditions that require it, a ureterocele should never be dealt with by open operation.

References.

[1] Bostroem: "*Beiträge zur pathologischen Anatomie der Nieren*", Number i, 1884; quoted by H. H. Young and D. M. Davis in "Young's Practice of Urology", Volume ii, page 30.

[2] P. Wagner: "*Angeborene Missbildungen der Nieren, Nierenbecken und Harnleiter*", *Handbuch der Urologie*, Volume ii, 1905; quoted by H. H. Young and D. M. Davis, *loco citato*.

[3] P. Poirier: "*Sur quelques phenomènes consecutifs aux injections ureterales*", *Comptes rendus de la Société de biologie*, Paris, Volume ix, 1891, page 585.

[4] C. M. Harpster, T. H. Brown and H. A. Delcher: "Abnormalities of the Kidney and Ureter", *Journal of Urology*, Volume viii, 1922, page 459.

[5] H. H. Young and D. M. Davis: "Young's Practice of Urology", Volume ii, page 46.

[6] Frank Hinman: "The Principles and Practice of Urology", 1935, page 450.

[7] J. C. Ainsworth-Davis: "Ureterocele: Some Observations Based on the Investigation and Treatment of Four Cases", *The British Journal of Surgery*, Volume xix, 1931-1932, page 548.

[8] Matthew White: "Ureterocele: Report on a Case of Ureterocele with Double Orifice", *British Journal of Urology*, Volume viii, 1936, page 266.

[9] Frederic E. B. Foley: "A New Method for Cystoscopic Surgical Treatment of Ureterocele", *Urologic and Cutaneous Review*, Volume xxxv, 1931, page 49.

A CASE OF INTRACRANIAL AEROCELE COMPLICATED BY STREPTOCOCCAL MENINGITIS.[1]

By Gilbert Phillips.

(From the Department of Neurosurgery, Royal Prince Alfred Hospital, Sydney.)

This case of intracranial aerocele, following a head injury, is of interest both because of spontaneous absorption of a large subdural collection of air and because of the rapid effect of "Prontosil" after the development of a streptococcal meningitis.

The patient was referred to the Royal Prince Alfred Hospital by Dr. Hill, of Coonabarabran, in March, 1938. He had fallen from the top of a tank stand, striking his head and rendering himself unconscious. On regaining consciousness, after a period which cannot be ascertained, but which was probably not more than half an hour, he had a profuse epistaxis and felt dizzy and confused. He went home to bed, where it was noticed after the cessation of the epistaxis that there was a profuse cerebro-spinal rhinorrhœa from the right nostril. This was much worse when the patient sat up.

At rest in bed, he recovered completely from the effects of the head injury with the exception that the rhinorrhœa continued and was particularly profuse when he sat up to get out of bed. He remained at home in this condition for two months, and in May, 1938, he commenced to suffer from headaches. It was then discovered that he had a mild pyrexia. He was sent down to the Royal Prince Alfred Hospital on June 9, 1938, with a diagnosis of post-traumatic meningitis.

On admission he complained of pain in the left eye and in the left side of his forehead, and he had a free flow of cerebro-spinal fluid escaping from the right nostril and running down the lower part of his face and chin on the right side. The right pupil was larger than the left and there was some weakness of the right side of the lower part of his face.

On examination it was found that he had diplopia on attempted convergence and disturbance of inward movement of the left eye. There was no nystagmus and slight ptosis was present on the right side.

There was some weakness of hand grip on the right side, but no disturbance of tendon reflexes in this extremity. The abdominal reflexes were present in all four quadrants. There was no disturbance of power, posture or tendon reflex action in the lower limbs. Plantar reflexes were flexor on each side. There was no disturbance of superficial or deep sensibility.

His temperature on admission was normal and remained so for one week.

X-ray examination of the skull on June 16, 1938, disclosed a very large subdural collection of air in the left frontal region, which increased in size until June 28, 1938 (see Figures I and II), when it measured 10 by 8 by 7 centimetres. Although this collection of air was so large, no abnormality was seen in the fundi, and subsequent lumbar puncture did not disclose any increase in cerebro-spinal fluid pressure. On June 17, 1938, his temperature rose to 101° F. and again to 100·4° F. on June 19, 1938.

At lumbar puncture on June 21, 1938, when his temperature was 101·6° F., the pressure was 90 millimetres and the fluid contained 616 cells per cubic millimetre, chiefly polymorphonuclear cells. A hæmolytic streptococcus was cultured from the fluid.

[1] Accepted for publication on December 8, 1938.

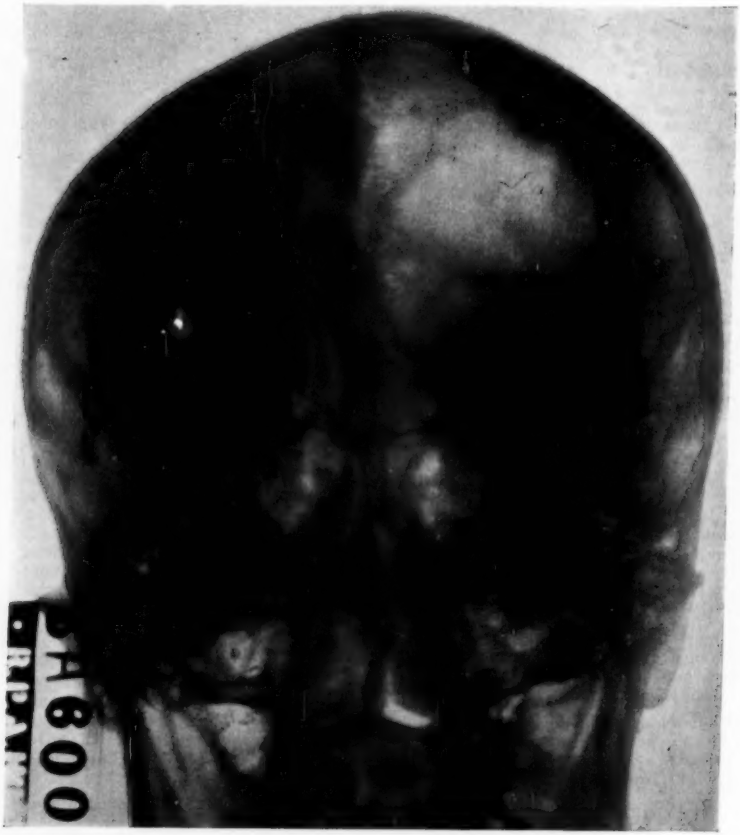
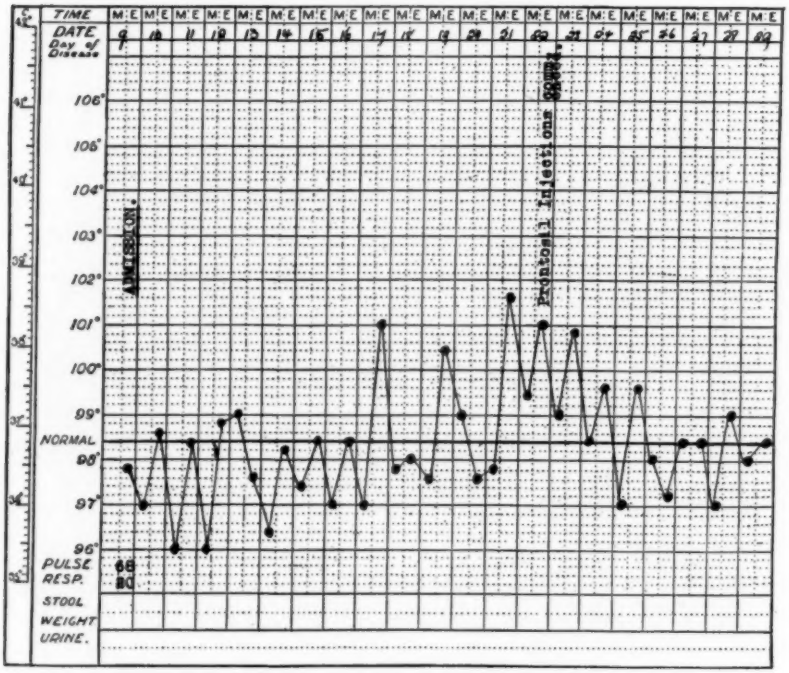


FIGURE I.

FIGURE II.

On June 22, 1938, "Prontosil" injections were commenced intramuscularly every six hours. His temperature gradually fell during the next three days, and on June 24, 1938, lumbar puncture showed the fluid to be still slightly turbid; the pressure was 90 millimetres, but 1,300 cells per cubic millimetre were present and were polymorphonuclear.

As shown in the accompanying chart, his temperature was normal on June 29, 1938, and a further lumbar puncture on June 30, 1938, yielded clear fluid under a pressure of 100 millimetres; the total protein content was 60 milligrammes *per centum* and the fluid contained 226 cells per cubic millimetre.



Operation to remove the aerocele and repair the dural fistula was considered inadvisable, not only because the aerocele was not producing any general signs of cerebral compression, but also because it was regarded as likely that the "Prontosil" had produced a condition of bacteriostasis, and that a recurrence of the meningitis might not be improbable. During the succeeding three months, four such recurrences took place, the temperature rising to 100° and 101° F. on each occasion and falling to normal within sixty hours on administration of "Prontosil". The last of these recurrences occurred from July 5 to 7, 1938, but he has been afebrile from that time up to the present.

Serial skiagrams (see Figures III and IV) taken on July 13 and 28, 1938, show a progressive decrease in size of the aerocele, and on August 24, 1938 (see Figure V), the aerocele had completely absorbed.

FIGURE III.

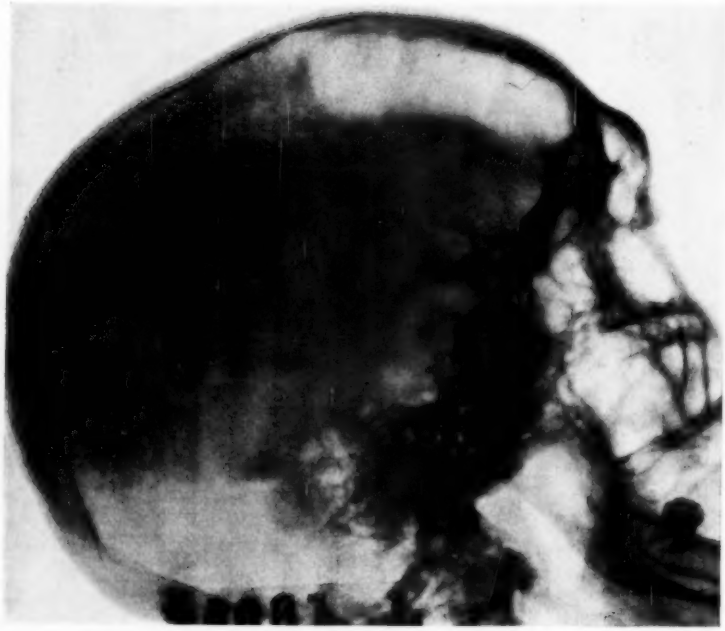


FIGURE IV.

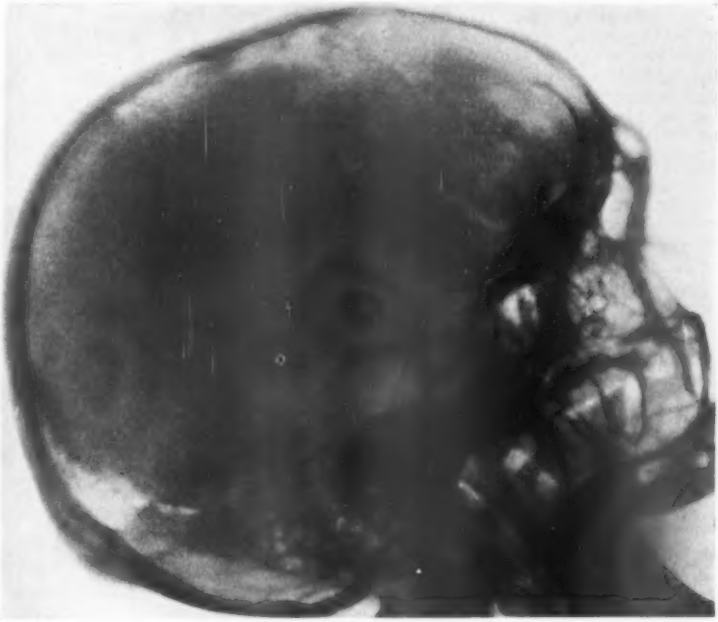


FIGURE V.

During July, August and September there was a progressive decrease in the amount of cerebro-spinal fluid rhinorrhœa. At first the fluid would drain away even when the patient was recumbent; later he would lose one or two ounces from the right nostril on sitting up; this amount decreased until he would only occasionally lose one or two drachms in the upright position. On October 17, 1938, the rhinorrhœa had ceased.

It is important to note that, in the presence of an intracranial aerocele, cerebro-spinal fluid rhinorrhœa from one nostril does not lateralize the collection of air. In this case, in the presence of a large aerocele on the left side, there was a profuse rhinorrhœa from the right nostril and at no time did any cerebro-spinal fluid escape from the left nostril.

Judging from this case it would seem desirable in other cases of intracranial aerocele, not complicated by streptococcal meningitis, not to operate in an attempt to remove the aerocele and close the dural fistula, unless there is some definite indication, such as papillœdema, until an opportunity had been given for the air to absorb and the fistula to close spontaneously.

Encephalography has been advocated in these cases to evacuate the aerocele when it is situated between the arachnoid and the dura. This procedure was contra-indicated in this case by the presence of a hæmolytic streptococcus, and must in any case involve the risk of aspirating infective material from the nasal passages through the dural fistula into the subarachnoid spaces.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in indian ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

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THE SURGEON AND THE WAR.

IN these days of totalitarian warfare, the sudden change from peace to war-time conditions raises innumerable problems to all sections of the community, but to none more than the medical profession. The large and constantly increasing part played by the medical services in the wars of this century has imposed on this army service greater and greater obligations. The maintenance of the strength of armies in the field through the prevention of disease and the collection, removal, distribution and care of the sick and wounded and the treatment of war injuries on a large scale, demand an organization of great complexity and of a very high degree of efficiency. In peace, such an organization is embodied in the permanent war forces of a country. But it must necessarily be of an embryonic character compared to the fully developed war-time body into which it quickly grows. This fully developed war-time medical service must include the best surgical ability available, and it must envisage methods to provide operative treatment at the earliest possible moment; that is, from a national standpoint not only must the wounded receive the most skilful surgical treatment, but it is one of the most important principles of modern war surgery that it should be instituted with the utmost promptitude.

In times of crisis, a considerable proportion of the surgically trained personnel must be taken from the civil population. The practitioner, however, must not forget that a knowledge of his own subject does not automatically make him fit to deal with all aspects of military surgery—a fallacy to which he may sometimes be liable, because of the individualistic attitude of mind to which he is almost inherent as a result of the training and practice of his profession. A short time spent in a military unit will soon convince him of the unsoundness of such a view, and demonstrate to him that, in war, the medical man must submit to the training and discipline of the permanent army medical forces.

There are two principal problems facing the military surgeon; many minor ones must, for the moment, be ignored. These are: (*a*) The organization and the administration of a large number of units, many of which come into being only as a result of the advent of hostilities. (*b*) The origination and development of certain aspects of war surgery which arise as a result of high specialization in casualties peculiar to modern warfare—surgery which is very different from anything encountered in civil practice.

In regard to the first problem, the question of organization, there is no need to make a statement here since very few surgeons have had sufficient training to allow of their taking over, without preparation, an administrative position. The very prevalent idea that a practitioner, who may be preeminent in his own branch of medicine, should be able forthwith to command a field ambulance, administer a hospital, organize a field hygiene section or a mobile hygiene or bacteriological laboratory, needs but little contemplation to demonstrate its absurdity. It is not uncommon to hear criticisms of the "red tape" of the military machine, or the "hide-bound" regulations. What is not appreciated, however, is that the machine is an extremely complicated mechanism which is the result of slow growth over many decades. This mechanism, like all complex mechanisms, is very delicate and, as such, will give satisfactory results only when its working is thoroughly understood: the intelligent and well informed can make it work efficiently and silently; the unintelligent and uninspired will cause it to move slowly and to creak. Ignorance is equivalent to the "spanner" which stops the machine, and the organization will go backward rather than forward. Unfortunately, not only does the particular piece of mechanism stop, but a number of related units also become obstructed. A thorough knowledge of the organization and of its relationship to other units is, therefore, obviously essential for any officer occupying an administrative position.

In regard to the second problem, the aspects of surgery peculiar to war, there are the technical difficulties in carrying out medical treatment, surgical procedures and pathological investigations under conditions vastly different from those of civil life. One has only to compare some of the French instruments, especially scalpels, with those of other nations to realize how

a country which has had numerous wars (as the French have had in North Africa) over the last few decades, will develop a special and superior armamentarium. The surgeon must also adapt himself to working with the range and varieties of instruments provided. There is no place for "fads" in the war machine: the problems of supply and transport are too great. That experience of war alone can give a reasonable appreciation of the methods of overcoming difficulties is shown by the diametrically opposed opinions regarding what equipment is desirable or necessary or even essential for various units, given by officers who worked during the last war and younger officers who did not. The way in which methods of treatment develop during active warfare is well shown in the elaboration of the procedure of wound excision, well-recognized by Larrey, Napoleon's great surgeon, and rediscovered in the Great War, and in the treatment of various bone and joint injuries, in plastic and chest surgery, in the application of blood transfusion, in the correct appreciation of gas infection, to say nothing of the innovations that have arisen in the Spanish war.

Although much experience was gained in the Great War, nevertheless it is to be expected that there will be new problems in the present struggle and that these will have to be met by keen, probably young minds, unfettered by preconceived ideas of what has been suitable in civil life—or even in past wars. From the solutions of such problems there can be little doubt that civil surgery will benefit—perhaps to as great a degree as in the last war, a degree that we are apt to forget; and thus it may be some consolation to the surgeon that he may take something with him out of the present crisis which he can apply for the benefit of humanity.

RUPERT M. DOWNES.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

THE EARLY STAGES OF RENAL TUBERCULOSIS.

Heim (Kiel): *"Nierentuberkulose mit besonderer Berücksichtigung des Frühstadiums"*, *Der Chirurg*, April 15, 1939.

HEIM's thesis is that renal tuberculosis is not so rare as is thought, but that the harmless nature of its early symptoms causes it to be missed. He quotes Clairmont as reporting that of 153 cases of renal tuberculosis occurring in patients admitted to hospital under his care, 79 were unrecognized and 50 were diagnosed as cystitis. Every acute cystitis which does not respond to treatment, every chronic recurring cystitis, and every case of chronic albuminuria, however slight, should be suspected. He quotes the history of a man who, on completing his military service, had to submit to a routine examination for admission to the customs service. Albuminuria was the sole positive finding, but animal inoculation with the urine revealed a tuberculous infection; a pyelogram revealed a left-sided renal infection, and at operation a severely infected kidney was removed.

In most cases, none the less, slight but classical symptoms are present. Bier's description to his students holds good: "In a patient who complains of being awakened suddenly out of sound sleep to pass urine, and in whom you find albumin and blood and pus cells in a urine which is acid in reaction, you may diagnose tuberculosis." Urine containing pus without any organisms is suspicious for tuberculosis, and should be investigated. The sediment of a twenty-four-hour specimen is required, and should be stained by Konrich's method[1] and also inoculated into guinea-pigs. Tuberculin injection is also used in the diagnosis.

Ascending infection is very rare, and the primary focus is usually a silent one, but expert examination of chest films and careful examination of the prostate and epididymes should never be omitted. The classical symptoms of advanced renal tuberculosis need no emphasis; but the early general symptoms of loss of energy, fatigue easily produced, sweating, loss of weight *et cetera*, which take the patient to the doctor, should make one think of tuberculosis, and a careful urinary examination may show the seat of it to be renal. In women, complaints of lower abdominal pain should turn one's thoughts to possible renal tuberculosis, and a urinary examination should be done before a retroverted uterus is credited as being the cause. A tender spot where the ureter crosses the pelvic brim is especially suspicious. In any nephritis, if renal tuberculosis is once suspected, an accurate diagnosis must be made as early as possible, so that the sound kidney may be protected from toxic effects and actual infection.

A little-known sign of early renal tuberculosis is the non-specific urethral discharge, and a urethral discharge not containing organisms, equally with urinary pus without organisms, must make the exclusion of renal tuberculosis a primary duty. This urethral discharge may be secondary to a cystitis, but in Heim's experience it may arise reflexly without any involvement of the bladder. It must also be borne in mind that the onset of renal tuberculosis may be acute and be accompanied by colicky pain, and any such case not otherwise explained must be suspected.

Cystoscopy in the early stages gives very little indication of the renal condition, and the same is true of dye excretion tests. Ureteric catheterization is the only safe method, the urine from each kidney being separately examined for bacilli. Heim does

[1] Konrich's method of staining tubercle bacilli is said by Baeckmann to be far more effective in displaying them than the Ziehl-Neelsen method. It consists in staining with carbol-fuchsin, decolorization with 10% sodium sulphite solution, washing first with 60% alcohol, then with water, and counterstaining with 0·2% aqueous malachite green.

not hesitate to do this at the first examination. He considers that the risk of carrying up an infection is negligible if careful technique is used, and he wishes to avoid the great delay involved in animal inoculations of urine first from the bladder and later from the renal pelvis.

Alterations in dye excretion appear only late in the disease, and it is a gross error to rely on it in the early stages. Similarly intravenous pyelograms are not sufficient for diagnosis. Retrograde pyelograms, on the other hand, are of prime importance. The nidus of infection is often in the fornix of the calyx, where it causes epithelial damage and breaks through into the pelvis. Flattening of the tips of the calyces, arched displacement of them and a blurring of their outlines are definite signs of tuberculous infection. A very quick pain response to injection of the contrast fluid indicates an irritability of the renal epithelium, similar to the irritability of the bladder. It is essentially an early symptom, but in itself gives no indication of the extent of the disease. The ulcerative type of tuberculosis tends to be limited to one or other pole, and special attention must be paid to the poles in the study of the pyelograms. This type is inclined to be unilateral and is particularly prone to secondary infection and abscess formation. The miliary type infects mainly the tips of the calyces, the infection remains pure, and has a much greater tendency to bilateral spread. Its more silent symptoms, its acute nature, and the bilateral tendency make its prognosis less encouraging than the other, and in the bilateral cases nephrectomy must be approached with great caution.

Should nephrectomy not be advisable, general treatment is not very hopeful. "Urotropin" makes the condition definitely worse. Sailer, of Budapest, has reported good results with the use of "Rubrophen" in surgical tuberculosis. "Rubrophen" is trimethoxydioxyoxotritan, and is said to have not only a good effect on the patient's general condition and to produce an increase in weight, but also a direct effect on the diseased tissues themselves. Any such new material would be worth trying in these distressing cases.

Renal tuberculosis is a disease which shows little tendency towards spontaneous healing or response to conservative measures. The earlier a unilateral tuberculous infection of the kidneys can be fully diagnosed and removed by operation, the better will be the percentage results.

ARTHUR E. BROWN.

REPORT ON A CASE OF COMPLETE COLECTOMY FOR ULCERATIVE COLITIS.

Dr. Wolfdietrich Weissenborn, Wiesbaden: *"Beitrag zur Entfernung des gasamten Dickdarmes bei chronischer ulceröser Colitis, unter besonderer Berücksichtigung eines Dauererfolges", Der Chirurg,* June 1, 1939.

DR. WEISSENBORN reports an interesting case of ulcerative colitis, in which the constant deterioration of the patient's condition forced him to undertake a radical removal of the entire large bowel. He writes: "It seems to me that more important than a mere technical description of the operation is the question of how such a patient gets on without his colon in later years." The report therefore includes a full investigation of the patient three years after his final operation.

The patient was a young man, eighteen years old on his first admission to hospital, and had already been three years under conservative treatments of various sorts. He was admitted to hospital with the idea of having a short-circuiting operation done to exclude the affected bowel. His general condition was extremely poor; his hæmoglobin value was 45% and red corpuscular count 2,200,000 per cubic millimetre. The Wassermann test produced no reaction, and the bacterial flora of the bowel did not contain any abnormal elements. Proctoscopy revealed ulcerative changes within thirty centimetres of the anus. X-ray appearances following an opaque enema seemed to suggest that the disease was largely limited to the bowel distal to the middle of the transverse colon.

In May, 1932, the first operation was done, an ileostomy under local anæsthesia being followed by irrigation of the bowel with various solutions. Neither diminution in the discharge of blood and pus nor lowering of the high temperature was observed, and after five months the proctoscope revealed no more improvement than did the clinical examination of the patient. After eleven months' treatment it was decided to remove the colon.

In April, 1933, the second operation was performed. The ileostomy opening was closed and a lower median incision was made. The whole large bowel, from ileo-caecal

junction to the rectum, was then seen to be equally affected by the disease, though the small bowel appeared to be quite normal. The sigmoid colon was cut across at the lower edge of its mesentery, and the distal stump was fixed outside the peritoneum. The sigmoid, descending colon and the distal half of the transverse colon were resected, the stump of the transverse colon being closed. The ileal fistula was reopened. This operation was followed by a large abdominal wall abscess some five weeks later, and by but little improvement in the patient's general condition. The discharge of blood and pus from the rectum continued unabated. There was, however, some slight general improvement.

In August, 1933, a third operation was performed through a right pararectal incision, the remainder of the transverse colon, along with the ascending colon, the caecum and five centimetres of the terminal part of the ileum being removed. It was very well tolerated, the post-operative course being only marked by various abdominal wall abscesses which had to be opened. Histological examination of the bowel removed at both of these operations revealed a chronic inflammatory condition of the mucosa and the submucosa, with small ulcerating defects in the mucosal surface. The division between diseased colon and healthy ileum was very well defined and sharp. Three months after this intervention the patient's condition was so much improved that he was able to get out of bed. He now complained of discomfort from the pressure of his colostomy apparatus on the anterior iliac spine, and his fourth operation was then carried out, the ileostomy opening being placed in the mid-line. General improvement continued, but rectal inspection showed that there was no such improvement in the local condition there. During the ensuing year the position varied, periods of good health alternating with periods of high fever and increased rectal discharge. Several times abscesses in the region round the anus and the scrotum had to be opened.

Finally, in January, 1935, the removal of the rectum had to be advised, and was carried out after removal of the coccyx, the anal sphincter and the whole remaining rectal stump being resected. This wound healed slowly, and the patient had an immediate gain in weight and in his hæmoglobin value and red blood cell count. All that was done subsequently to this was to move the ileostomy opening again to his left side, at the patient's own request.

The course of the disease so far indicated that it was possible in such a desperate case to remove the whole of a patient's large bowel, and that no method short of doing this would have cured the trouble. The next question was: What sort of a life would be possible to a young man after such an operation? Three years after his last operation he submitted himself to a complete investigation. He reported that his health had been entirely satisfactory and his appetite good, with no need to control his diet in any way. Digestion was normal, and he had on the average two bowel actions daily, the stools being of normal consistency and colour. He was able to recognize the imminence of an action from the ileostomy and, by lying across the closet seat, to keep himself free from soiling. His urine output was normal. He worked daily and was earning money by his trade. He had even tried to take up sports, but the "colostomy bag" prevented him from doing this. He participated in the dances and amusements of other young men, became engaged and was looking forward to being married.

An objective examination showed him to be in excellent health. There was no inflammation round the ileostomy opening, the orifice of which admitted about a thumb, and was controlled by a device of his own planning. X-ray examination of the bowel after an opaque meal was very interesting, for although the radiologist had been informed of the previous history, he found it hard to believe that the films were those of ileum and not of ascending colon. It was evident that the lower part of the ileum had taken over in compensatory fashion the functions of the lost colon. Not only the films but the character and constitution of the stools illustrate this fact. Analysis of the stools reveals them to be of entirely normal constitution, and their bacteriological examination revealed a typical large-bowel flora. The blood on examination was normal. The hæmoglobin value was 92%, the red cells numbered 4,540,000 and the leucocytes 7,800 per cubic millimetre. Polymorphonuclear cells numbered 57%, band forms 2%, lymphocytes 21%, monocytes 2%, eosinophile cells 15%, basophile cells 3% and platelets 331,420 per cubic millimetre. Sedimentation rate was normal. The figure obtained on estimation of the blood sugar was 113 and of the blood protein nitrogen 26·6; the blood calcium content was 13·2%. Exhaustive analyses of the urine for all usual and possible constituents gave an entirely normal finding, except in one respect. The calcium content of the urine was enormously increased from the usual 0·8% to 164·22 milligrammes

per centum. Calcium excretion is in normal circumstances a function of the large bowel, and in this case the kidneys had taken over this function.

On the whole, the case demonstrates that a complete colectomy in man is possible both for the surgeon and the patient. But Weissenborn ends justly with the remark that such an operation would obviously be undertaken only in very exceptional circumstances.

ARTHUR E. BROWN.

Reviews.

A Short Textbook of Surgery. By C. F. W. ILLINGWORTH, M.D., F.R.C.S.; Second Edition; 1939. London: J. and A. Churchill Limited. Medium 8vo, pp. 710, with 8 plates and 179 text-figures. Price: 21s.

THAT a second edition of this volume has become necessary within a year shows the need for this type of "shorter surgery". The author has avoided the tendency to enlarge second editions; in fact, this volume contains fewer pages than its predecessor.

New matter introduced concerns the use of sulphanilamide in gonorrhœa and other acute infections, the value of sympathectomy in essential hypertension, and the use of vitamin *K* in jaundice.

A valuable new sections deals with the increasingly important subject of water balance in the surgical patient. The increase in parenteral therapy has shown that there is greater danger of over-dosage than of under-dosage when this method is used by the inexperienced.

We still think that the book is not quite detailed enough for honours students.

The Surgery of the Alimentary Tract. By SIR HUGH DEVINE, M.S., F.R.A.C.S., F.A.C.S.; 1940. Bristol: John Wright and Sons, Limited. Australia: Butterworth and Company (Australia) Limited. Demy 8vo, pp. 1057, with 690 illustrations, some in colour. Price: 70s.

THIS book, by an experienced Australian surgeon, is both interesting and original, and, though at times provocative, it is one of the best books on the subject that we have read. It might be said that this is a book with a personality, as it truly reflects the personality of a keen practical surgeon who has shown himself a master of his art. In these days of dull medical books, so many of which merely repeat other writers, it is very refreshing and stimulating to meet new suggestions and ideas which are often presented in quite a novel fashion.

The author does not attempt to cover the whole field of ætiology and pathology, which, as he states, is well done in numerous other books and articles; but gives his own views, and these, though at times mildly unorthodox, are often original and stimulating. He supplies many interesting illustrative cases, and attempts, often with success, to correlate the clinical picture with the operative findings. We find many novel features in this work, such as the introduction of chapters dealing with imaginary consultations on the interpretation of gastric lesions by radiography (this should be read by all radiologists), while throughout there is reflected the attitude of the practising clinical surgeon, who has worried through many difficult problems himself and who here sets out his experiences and deductions for all to read.

The book is packed with practical points in diagnosis and technique, and is a real contribution to surgery. One feels that the author has at times laid too much emphasis on rather uninteresting and relatively inaccessible continental contributions without paying enough regard to the advances made by British and American surgeons.

The book has a wealth of illustrations, the majority of which are very good and clearly demonstrate something worth while; at the same time there are a few unnecessary repetitions and a few rather poor reproductions of radiographs.

The early part of the book deals with the clinical aspects of dyspepsia, one of the commonest and the most difficult of all symptoms to interpret. The author asks

pertinent questions as to the cause of pain and its relief by various procedures; he records good illustrative cases, answers most of the problems by a correlation of symptoms with pathology, and, although at times rather dogmatic, his handling of this subject is excellent and should be of value to all clinicians.

The subject of dyspepsia has interested the author all his life, and there is packed away in these chapters a wealth of clinical experience. The section dealing with the reflex dyspepsias, chronic gastritis, and chronic gastric ulcer are very good and illustrate well the rather novel methods of approach to clinical problems which the writer uses throughout the book. The section on jejunal ulcer is very good indeed—one of the best studies extant; it is well illustrated with cases and pictures and contains a number of practical technical tips. In dealing with cancer of the stomach the author rightly emphasizes the protean nature of the clinical picture, and although there is nothing new, this section is dealt with in an attractive manner. In the treatment of profuse hæmatemesis from erosion of a large vessel he favours partial gastrectomy rather than ligation of the various vessels, but this section lacks the stimulating dogmatism of some of the other sections. In the management of this vexed problem we rather looked for more clear-cut guidance in regard to details.

As is to be expected from one who has added so much to operative technique and who has, during his surgical life, shown a surprising mechanical originality, the sections dealing with operative technique are very good. The author's well-known fixed retractor is the centre piece of practically all the technical procedures, and in his hands it is an astonishingly useful instrument. The small drawings and photographs illustrating operations are very clear and, unlike many surgeons, he has given very full details of his work. This section, though of interest to all surgeons, should be particularly valuable to young surgeons, as there is a wealth of illustrations, and in such operations as partial gastrectomy nothing is left out. The drawings of the method of closure of the duodenum are especially good.

There follow special chapters on the management of abdominal adhesions, diaphragmatic hernia, splenectomy and hydatid disease of the liver, in all of which the author, as he states in the preface, gives his own experiences and technique and does not attempt to deal with all methods. His predilection for the paramedian incision rather than the transpleural route as an approach to the upper surface of the liver is shared by many surgeons.

Diseases of the gall-bladder are dealt with mainly from the clinical aspect, and this is an interesting and instructive section. The author steers a middle course in regard to the time for operation on acute cholecystitis, although he does not emphasize sufficiently the importance of severe liver damage in many of these cases as a factor in prognosis.

The section dealing with diseases of the pancreas is rather disappointing, and the author fails to give any recognition to the remarkable work on pancreatitis by Archibald, who added more to our knowledge of this disease than any other investigator. One is left in doubt as to whether the author himself really advocates conservative, non-operative treatment in acute pancreatitis or not.

The sections on post-operative complications, their division into three chronological groups, although there is nothing new or original in them, are well arranged and valuable. They would have been more valuable if more detail had been given and if more definite statements had been made as to the exact procedure advised by the author himself; we should have expected rather more credit to be given to Wangensteen and to Wood for their very fine work on the problems of fluid balance. It is, too, rather a pity to see the use of intravenous gum arabic solution, a now discredited method, advocated in shock.

Two very useful chapters follow on the procedures advised in such surgical emergencies as perforation and intestinal obstruction; both are well done and full of sound advice and suggestions culled from a great experience.

The chapter on appendicitis is one of the best in the book; the author deals with the subject, not along ordinary text-book lines, but discusses in full all the difficulties and problems which an experienced surgeon has met with during a long practice. The same is true of the section on diverticulitis, where his successes and failures are recorded in his attempt to shed some light on, and to provide some help in, the management of what are perhaps some of the most difficult problems in abdominal surgery.

The last chapters are devoted to the surgery of the colon and rectum, and in all the author maintains the same method of correlation between clinical history and

pathology; in all he is original and even at times frankly experimental, while at others he is so provocative that no surgeon can fail to profit by reading these well illustrated sections. Some will disagree, but all will, we are sure, find practical points and suggestions for the improvement of what is always rather difficult surgery.

Altogether the book is a great one and a credit to Australian surgery. The art and ideas of Devine will not die because, unlike many skilled surgeons, he has made available all his knowledge for the study and profit of his successors; and in so doing he has put into practice that Hippocratic ideal. As the writer states, this is not a text book suitable for undergraduates, as it presupposes a fairly wide knowledge of the subject; but it is essential for Australian post-graduates and should also be in the hands of all surgeons, physicians and radiologists whose work brings them in contact with diseases of the alimentary canal.

H. R. DEW.

The Diagnosis and Treatment of Diseases of the Esophagus. By PORTER P. VINSON, B.S., M.D., F.A.C.P.; 1940. Springfield: C. C. Thomas. London: Baillière, Tindall and Cox. Medium 8vo, pp. 224, with illustrations. Price: $4.00 net.

IN this monograph is brought together, by one whose experience in this field is unrivalled, all that most surgeons need to know to enable them to make a diagnosis for and correctly advise or treat patients suffering from any of the recognized diseases of the œsophagus. The book is well produced and easily read, and the many excellent illustrations add greatly to its attractiveness and value. The information and the opinions expressed in the book are based on the study during nineteen years of approximately three thousand patients with some abnormality of the œsophagus, and the practical soundness of the conclusions reached is apparent on every page.

Although for exact diagnosis radiological examination alone or combined with examination by the œsophagoscope is required in most cases, the author shows how an accurate interpretation of the history and clinical signs will nearly always enable the medical attendant to make a reasonable provisional diagnosis.

Neuroses of swallowing are common, but real difficulty in swallowing is nearly always the result of organic disease, and if this fact were more widely recognized, the chance of early diagnosis and successful surgical extirpation of cancer would be much better. In the author's experience of patients with carcinoma of the œsophagus, symptoms have been present for an average of seven months before diagnosis was made.

Hydrophthalmia or Congenital Glaucoma: Its Causes, Treatment and Outlook. By J. RINGLAND ANDERSON, M.C., M.D., B.S., F.R.C.S., F.R.A.C.S., D.O.M.S., with a foreword by SIR J. H. PARSONS, C.B.E., D.Sc., F.R.C.S., F.R.S.; 1939. Cambridge: The University Press (for *The British Journal of Ophthalmology*). Royal 8vo, pp. 397, with illustrations. Price: 25s. net.

THIS work by J. Ringland Anderson, of Melbourne, is an outstanding volume. Eight years ago Anderson published, also under the auspices of *The British Journal of Ophthalmology*, a monograph on retinal detachments, and after summarizing the theories of causation he detailed the results of treatment up to the beginning of the Gonin era. In this, his second monograph on hydrophthalmia, he has also surveyed the various methods of the treatment of that rare disease (which has been familiarly known for many years as buphthalmia).

The first chapter deals with the theories and facts regarding ætiology, and it draws conclusions from reported cases. The author sent 874 *questionnaires* to 346 oculists living in thirty-two different countries, and in all 111 completed forms were returned, but only nine of these came from Australia, and throughout the book there is little reference to the prevalence of this devastating disease throughout this Commonwealth. This is to be regretted. It is apparent that inheritance in hydrophthalmia is both rare and obscure; and in dealing with the influence of consanguinity, Anderson states that it appears to be of little significance.

The next chapter on "Differential Diagnosis" is of extreme importance. The author discusses differential diagnosis and gives a table showing the various characteristics of megalocornea compared with those of buphthalmos. He points out the high instance

of heredity (in apparently a sex-linked form) in megalocornea as compared with that found in buphthalmos.

In the third chapter the structure and development of the involved tissues are dealt with in detail, and chapter four is devoted to pathology. Although Anderson has made an extensive survey of the literature, he has been unable to show much world progress in the pathology of this disease since Sir John Parsons wrote his four volumes on the pathology of the eye over thirty years ago.

He summarizes the pathological conditions in the anterior chamber in the following four points: (*a*) an absence in Schlemm's canal; (*b*) a persistent or undue development of the meshwork of the angle; (*c*) a peripheral union of the iris and the cornea, which may be due to lack of differentiation or post-inflammatory synechiæ; (*d*) central corneal opacities with adhesions to the iris or the pupillary membrane. Here is a branch of pathological research open for further investigation. Unfortunately the author does not touch fully on the numerous congenital anomalies of the eye which have been reported in association with congenital glaucoma; nor does he more than mention associated congenital abnormalities in other parts of the body.

The fifth chapter considers the pathogenesis and the theories of origin of hydrophthalmia. Anderson here makes some mention of the possibility of birth injuries to the eye in buphthalmos. We think that in the light of J. Bruce Hamilton's article in the *Transactions of the Ophthalmological Society of Australia*, Volume i, on the effects of birth injuries to the eye and its appendages, this aspect of hydrophthalmia could be investigated further and might produce some fruitful results.

In the sixth chapter on "Treatment" we read a thorough discussion of the various effective and non-effective attempts to arrest hypertension. The outlook for the curative treatment of buphthalmos does not appear happy. Nevertheless, it is apparent that fistulization by one means or another (when myotics have failed) is the best approach. However, Anderson points out that success from any operative procedure almost vanishes after the first year of life, the exception probably being mild cases or those that are arrested spontaneously.

In the eighth chapter on prognosis it is clearly pointed out that the more advanced the hydrophthalmia, the worse the result of operative interference. In the last chapter on "General Reflections" the author indicates the apparent hopelessness of the position in view of our present limited knowledge.

We must congratulate Dr. Ringland-Anderson and his associates for this truly excellent monograph. The work will remain a standard of reference on the subject for at least the next twenty-five years in British, American and Australian medical literature alike. We cannot too warmly commend this monograph to any ophthalmologist interested in the fascinating features of congenital diseases of the eye.

Endocrine Gynæcology. By E. C. HAMBLEN, B.S., M.D., F.A.C.S., with a foreword by J. D. COLLIP, M.D.; 1939. Springfield: C. C. Thomas. London: Baillière, Tindall and Cox. Royal 8vo, pp. 481, with illustrations. Price: $5.50 net.

THIS monograph presents an excellent review of present knowledge concerning sex hormones, sex physiology and endocrinopathic gynæcology. The author states that the book is intended not so much for the specialist gynæcologist and endocrinologist as for the general practitioner who has not had the time to keep pace with the voluminous literature on this subject. It is not certain but that its very completeness may defeat this object. The presentation of a mass of extremely involved and at times conflicting data is apt to confuse a reader not equipped with a reasonably detailed knowledge of the fundamentals of the subject under discussion.

In the first section of the book a more or less condensed account is given of the pharmacological properties and therapeutic applications of the various sex-endocrine principles. Unfortunately, many of the commercial preparations discussed are of American origin and will be unfamiliar to the Australian reader. However, the same type of criticism may be made in America concerning English publications that neglect mention of any but English products. The clinician must be considerably confused by this multiplicity of nomenclature in respect of sex hormones, and it is time that steps were taken to simplify and clarify matters. Amongst œstrogenic substances, the present book quotes "Emmenin", "Theelin", "Progynon" and "Amniotin", whilst a recent English publication refers to "Œstroform", "Di-menformon", "Lutocyclin" *et cetera*. It would be quite practical to adopt a common name for hormones possessing the same pharmacological properties and then to state the maker's name.

In discussing androgenic substances, the author makes an excellent point in indicating that their suggested therapeutic applications are in many cases inferential and should not be taken as clinical applications. In a sense this applies to all of the hormones, and perusal of many of the brochures issued by commercial firms is apt to make one forget this fact.

The section on "gynecic" physiology is well developed, but exception must be made to that portion dealing with the actual cause of menstrual bleeding. Admittedly the question is highly controversial, chiefly with regard to whether the phenomenon is due to œstrin deficiency or progestin deprivation. The author gives the impression that he considers œstrin diminution or a qualitative alteration in its metabolism as the most likely cause of bleeding. In our opinion, the author, in discussing the various experimental findings, has failed to take into account the important interrelationship between œstrin and progestin in the causation of menstruation.

The last section of the book, dealing with endocrinopathic gynæcology, has an excellent chapter on diagnostic procedures, but pride of place must be taken by the portion in which attempts to alter pathological changes in the ovary by means of endocrine therapy are discussed. The author has the good fortune to work in close cooperation with a very efficient laboratory and his opinions, based on the correlation of clinical and laboratory findings, must be treated with considerable respect. The remarks on the results of endocrine treatment of functional menstrual disorders are highly provocative and stimulating. It is considered that no evidence exists to show that follicle stimulation, ovulation or formation of *corpora lutea* in the human ovary results from treatment with gonadotropes prepared from pregnancy urine or placental tissue. These conclusions are based on observations on fifty-seven patients in whom direct inspection of the ovaries was possible during treatment with gonadotropic preparations. The author considers that there is one group of patients upon which the gonadotropes have specific effects, namely, those patients who have menorrhagia with mixed endometria. On the other hand, it is believed that the "hæmostatic action" observed by many clinicians is non-specific, and, in agreement with Jeffcoate, he thinks that just as good, if not better, results could be obtained with oxytocic drugs, insulin, seakale or even relatively inactive hormone preparations.

In conclusion, the work represents a most successful attempt to review in a systematic manner a very wide field of experimental and clinical investigations. The many illustrations are admirable; a good bibliography and index are added, as well as a foreword by J. B. Collip.

Minor Surgery. By R. J. McNEILL LOVE, M.S., F.R.C.S.; 1940. London: H. K. Lewis and Company Limited. Crown 8vo, pp. 378, with 155 illustrations. Price: 12s. 6d. net.

THE author disarms criticism of this little volume by declaring, in the preface, the difficulty of defining "minor surgery". Admitting this difficulty, one asks for whom is the book written. The author states it is for house surgeons and practitioners, but it is certainly not full enough for house surgeons, and would seem to be more suitable as a primer for a student doing "casualty" or surgical dressership.

The subject-matter is concisely and clearly set out, and the illustrations are well selected. Besides surgery, there are sections on the eye, the ear and anæsthetics.

Treatment of Some Common Diseases (Medical and Surgical). By Various Authors, edited by T. ROWLAND HILL, M.D., M.R.C.P.; 1939. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 412, with illustrations. Price: 15s. net.

THIS is a most valuable series of monographs written by a number of well-known authors, all of whom have the privilege of being members of the honorary staff of the Southend General Hospital. Included in the list of authors are T. Rowland Hill, Wilfred Trotter, Sir W. I. de C. Wheeler, and William Evans. The editor claims that it has been his object "to produce a volume covering a considerable range of medical and surgical subjects that would prove of value to medical men in active clinical practice and, in particular, to general practitioners". This endeavour has been successful. A high standard has been maintained throughout. Each author has obviously enjoyed the writing of his section, and speaks as a master of his own particular realm. William Evans begins with a discussion on the treatment of *angina pectoris*, and shows the relative merits of the many drugs now used by cardiologists.

This chapter is of particular interest, as it demonstrates the value of well constructed charts in showing the results of treatment.

On the surgical side, Trotter discusses the early surgical treatment of malignant disease of the pharynx, and considers that, if the surgeon is given a chance of dealing with early cases, the results should be comparable with those obtainable in any form of malignant disease.

Wheeler discusses the treatment of enlargement of the prostate gland. He gives prominence to the Harris operation. He considers that it is "an attractive operation not only to those engaged exclusively in the surgery of the genito-urinary tract, but also to those who are accustomed to deal with plastic operations on the bile ducts; to those who repair cleft palates and perform the many plastic procedures with which the general surgeon is confronted as a daily routine". After discussing the necessity for team work and skilled nursing, he concludes by stating that, if conditions are ideal, the operation is strongly recommended.

The book is well illustrated and the publishers are to be congratulated.

The Clinical Diagnosis of Swellings. By C. E. CORRIGAN, B.A., M.D., F.R.C.S.; 1938. London: Baillière, Tindall and Cox. Royal 8vo, pp. 313, with 120 figures. Price: 18s. net.

THIS book gives a comprehensive and lucid account of the clinical diagnosis of swellings. Two-thirds of the work are devoted to a consideration of diagnosis according to pathological type; and included therein are inflammatory and granulomatous masses, tumours, cysts, ulcers, enlarged lymph glands and pulsating swellings. The remainder of the discussion is concerned with the problems of diagnosis according to anatomical situation and includes descriptions of hernia and of swellings of the neck, breast, abdomen, inguino-scrotal region and joints.

In the majority of works of this kind much space is occupied by descriptions of the methods of demonstration of clinical signs. This, however, has not been stressed by the author to any great extent. He has rather emphasized the explanation of clinical signs in terms of the pathological findings, and this, we think, is the best feature of the book. In this way it will be of particular value to the final year or post-graduate student rather than to the student who is entering upon hospital practice.

The text has been enhanced by some excellent line diagrams and classifications which should be of use both to student and teacher alike.

We think that the author has achieved his purpose, as stated in the preface, to produce a book in which is presented a simple, lucid, practical method of investigating swellings in clinical practice.

Orthopædic Operations: Indications, Technique, End Results. By A. STEINDLER; 1940. Springfield: C. C. Thomas. London: Baillière, Tindall and Cox. Imperial 8vo, pp. 766, with 865 illustrations. Price: $9.00 net.

THIS important book is the final account of the author's experience of various orthopædic operations, and with few exceptions only the methods practised by the author are described. Nearly every operation description is accompanied by splendid illustrations of the various steps. The writer justifies most of the important operations he recommends by informing us of the number of such operations he has performed and the end-results thereof. His statistics are very interesting and important. Not only are sound orthopædic operations described step by step, but accounts of modern amputations, operations on fractures, a very full account of repair of tendons of fingers with statistics, skin plastic operations, an account of operations for disabilities due to nerve disorders *et cetera*, are included. No important orthopædic subject appears to have been left out, and even coccygectomy, resection of Baker's cysts, and other operations not usually described, are included with illustrations. Indications for all the operations are concisely outlined. The whole presentation is orderly; the book is clearly written, abundantly paragraphed and fully indexed. It is a balanced account of the indications, details and results of modern orthopædic operations by an orthopædic authority and the book will be valued by all surgeons of experience and be a handbook of knowledge and guidance for the orthopædic graduate.

Proceedings of the Royal Australasian College of Surgeons.

ANNUAL REPORT OF THE COUNCIL.

THE following is the annual report from the Council to Fellows.

The Council of the College takes pleasure in submitting to Fellows the following statement recording College activities during the past twelve months.

Meetings of the Executive Committee.

The Executive Committee has met on 18 occasions. The attendances were as follows:

Sir Hugh Devine	18	W. A. Hailes (appointed Registrar to the Australian Board of Censors, July 26, 1939)	9
Sir Alan Newton	17	Julian Smith, junior (now on Active Service)	9
Sir Robert Wade	2	G. R. A. Syme	16
Balcombe Quick	16		
T. E. Victor Hurley	11		
A. L. Kenny	18		

Meetings of the Council.

Three meetings of the Council of the College have been held since the twelfth annual general meeting. The attendances were as follows:

Sir Hugh Devine	3	George Bell	2
Sir Alan Newton	3	T. E. Victor Hurley	2
Sir Henry Newland	1	A. L. Kenny	2
Sir Robert Wade	1	H. R. G. Poate	2
E. D. Ahern	2	Balcombe Quick	3
W. C. McCaw (deputy for F. Gordon Bell)	1		

Number of Fellows.

The College has a fellowship of 661. This figure is made up as follows:

Honorary Fellows	17
Overseas Fellows	15
New South Wales Fellows	184
New Zealand Fellows	134
Queensland Fellows	48
South Australian Fellows	47
Tasmanian Fellows	13
Victorian Fellows	178
Western Australian Fellows	25
	661

Death of a Vice-President.

It was with very great regret that members of the Council learned of the death of their colleague, the late Sir John McKelvey, Kt. The College has suffered a great loss as the result of the untimely death of Sir John McKelvey, because not only did he serve the College as a member of its Council, but also as a member of the Australian

Board of Censors, having on occasions acted as Chairman of the Board during the unavoidable absence of the Censor-in-Chief.

Death of an Honorary Fellow.

The Council desires to place on record its regret at the death of one of the Honorary Fellows of the College, the late C. H. Fagge, M.S., F.R.C.S., F.R.A.C.S. (Hon.). During his visit to Australia in 1932, the late Mr. Fagge made many friends, and it will be remembered that, as the representative of the Royal College of Surgeons of England, he came to Australia for the express purpose of presenting the College Mace, a gift from the Council of the Royal College of Surgeons of England to the Council of the Royal Australasian College of Surgeons. While in Australia he delivered the first George Adlington Syme Oration.

Deaths of Fellows.

The Council regrets to report the death of the following Fellows: W. C. Mansfield, New South Wales; Thomas Murphy, Victoria; Aeneas McDonnell, Queensland; Edward Ryan, Victoria; F. H. Vivian Voss, New South Wales.

Honours.

The President of the College, Sir Hugh Devine, Kt., has been honoured with the Honorary Fellowship of the Royal College of Surgeons of England.

Charter.

Advice has been received that His Majesty King George VI has issued an Order-in-Council to the effect that petitions for the grant of Royal Charters will not be received during the period of the war. This decision means that, although the petition lodged by the College was at the office of the Privy Council on the date on which the Order-in-Council was issued, it cannot now be considered until after the cessation of hostilities.

Gordon Craig Library.

The Gordon Craig Library continues to render a valuable service to Fellows of the College. The Council has also arranged that the library facilities might be placed at the disposal of members of the Australian Society of Anæsthetists and the Ophthalmological Society of Australia (British Medical Association), on terms agreed upon between the College and these two bodies.

The Council desires to place on record its gratitude to the following donors of books to the Gordon Craig Library: Sir Hugh Devine, Sir Alan Newton, J. Ringland Anderson, W. M. Box, R. Graham Brown, D. G. Carruthers, J. Bruce Hamilton, G. H. Hogg, W. Kent Hughes, A. L. Kenny, E. S. J. King, Robert Fowler, J. Thomson Tait, Dr. R. Andrew, Dr. Louis K. Gaggenheim, Dr. Geoffrey Kaye, Dr. J. W. Kennedy (Philadelphia), Dr. A. W. Hutson, Dr. C. V. MacKay, Dr. F. A. Wood, Dr. A. E. Rowden White, Ophthalmological Society of Australia (British Medical Association), University of Pennsylvania.

Gordon Craig Educational and Research Scholarships.

The Council has decided that, during the period of the war, no grants for research or educational scholarships shall be made from the income arising from the Gordon Craig Endowment.

Syme Scholarship.

Dr. Robert S. Lawson was appointed Syme Scholar for 1939-1940. The subject for the scholarship is: "The Results of Operations for Peptic Ulcers."

Journal.

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY is proving a popular medium for the publication of surgical papers. A large stock of unpublished material is at present on hand, and the Executive Committee of the Council has from time to time given consideration to the problem of publishing material received promptly. In an endeavour to reduce the stock of unpublished material on hand, an additional (seventh) forme

has for some time been published. The publication of this additional forme will be continued at least until the end of 1940.

Primary Fellowship Examination of the Royal College of Surgeons of England.

Advice has been received from the Council of the Royal College of Surgeons of England to the effect that the Primary Fellowship Examination which was to have been held in Australasia in 1941 will now be abandoned. It is most unlikely that a Primary Fellowship Examination will be held in Australasia during the period of the war.

Ryan Scholarships.

The examinations for the Michael and J. P. Ryan Scholarships in Surgery were held in December, 1939. The results were as follows: Michael Ryan Scholarship in Surgery, J. R. McCoy; J. P. Ryan Scholarship in Surgery, J. B. Curtis.

Meetings Arranged by the State and Dominion Committees.

During the twelve months the following meetings have been arranged by the various State and Dominion Committees:

New South Wales.—Sydney: Evening meeting, March 29, 1939. Sydney: Evening meeting, June 28, 1939. Orange: Annual meeting, October, 1939.

New Zealand.—Dunedin: Annual meeting August 30 and 31, 1939.

Queensland.—Brisbane: Annual meeting, August, 1939.

South Australia.—Adelaide: Annual meeting, August 30 and 31, 1939. Adelaide: Clinical meeting, February 14, 1940.

Tasmania.—Hobart: Annual meeting, November 17 and 18, 1939.

Victoria.—Ballarat: Half-day and evening meetings, May 27, 1939. Melbourne: Demonstrations at the departments of physiology and biochemistry of the University of Melbourne, September 1, 1939; half-day meeting, St. Vincent's Hospital, November 17, 1939.

Evening Meetings.

The following evening meetings have been held at the College:

July 21, 1939.—Film evening.
September 1, 1939.—C. Gordon Shaw: "The Surgery of the Knee."
October 11, 1939.—Fay Maclure: "Osteomyelitis."
October 12, 1939.—T. G. Swinburne: "Surgery of Petrositis."
November 27, 1939.—J. Thomson Tait: "Carcinoma of the Bladder."
November 30, 1939.—J. M. Buchanan: "Surgery of Gynæcological Malignancy."
December 13, 1939.—Kevin J. O'Day: "Exophthalmos."

Jacksonian Prize.

It was with very great pleasure that the Council learned that one of the Fellows of the College, E. S. J. King, had been successful in winning the Jacksonian Prize for the third time. The Council feels that E. S. J. King has brought not only great honour to the medical profession in Australia, but also to the Royal Australasian College of Surgeons.

Joint Committee of the Councils of the Royal Australasian College of Surgeons and the Royal Australasian College of Physicians.

At a combined meeting of the Councils of the Royal Australasian College of Surgeons and the Royal Australasian College of Physicians, held in Sydney in September, 1939, a joint committee of the Councils of both Colleges was formed for the purpose of dealing with matters of common interest to both bodies. Regular meetings of the representatives on this committee resident in each State have been held, and a great deal of work in the interests of Fellows and Members of both Colleges has been carried out. This work concerned particularly the activities of both Colleges during time of war, and as a result of the deliberations of the joint committee, schemes for the protection of the incomes of consultants and specialists absent on active service were devised. These schemes have been successfully launched in New South Wales, Victoria and South Australia. In addition, the committee has made representations to the Commonwealth Government and to the Department of the Army on matters which

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.

Balance Sheet as at January 31, 1940.

LIABILITIES.

	£	s.	d.	£	s.	d.
Sundry Creditors				20	16	0
Subscriptions Paid in Advance				42	3	0
Examination Expenses Reserve Account				401	18	7
Prince Henry's Hospital, Melbourne—Post-Graduate Teaching Fund				10	10	0
Gift Account				1,538	16	9
Syme Memorial Endowment Fund	2,511	0	11			
Syme Memorial Endowment Income Account	162	7	10			
				2,673	8	9
T. F. Ryan Endowment Fund	5,100	0	0			
Michael Ryan Scholarship Income Account	19	5	5			
J. P. Ryan Scholarship Income Account	19	5	6			
				5,138	10	11
Surplus Account—						
Balance, January 31, 1939	20,857	15	7			
Add Surplus Revenue for the year ended January 31, 1940	715	4	11			
				21,573	0	6
Craig Endowment Fund (as per separate Balance Sheet)				60,703	6	6
				£92,102	11	0

ASSETS.

	£	s.	d.	£	s.	d.
Cash on Hand				10	0	0
The Union Bank of Australia Limited—Balance at Credit				330	7	5
Advances to Honorary Secretaries				80	0	0
Investments—						
Syme Memorial Endowment	2,673	8	9			
T. F. Ryan Endowment	5,138	10	11			
General	6,275	5	1			
				14,087	4	9
Subscriptions in Arrear				168	0	0
Sundry Debtors				32	12	11
Land and Buildings—at Cost				13,960	19	5
Furniture and Fittings—at cost—*less* depreciation				2,230	0	0
Great Mace—at valuation				500	0	0
Craig Endowment Assets (as per separate Balance Sheet)				60,703	6	6
				£92,102	11	0

H. B. DEVINE, President.
BALCOMBE QUICK, Honorary Treasurer.
H. G. WHEELER, Secretary.

Audited and found correct.
YOUNG AND OUTHWAITE,
Chartered Accountants (Australia),
368 Collins Street, Melbourne.
February 12, 1940.

CRAIG ENDOWMENT.

Balance Sheet as at January 31, 1940.

LIABILITIES.

	£	s.	d.	£	s.	d
Endowment				57,108	3	3
Revenue Account—						
Balance, January 31, 1939	2,863	0	3			
Add Surplus for the year ended January 31, 1940	2,367	0	5			
	5,230	0	8			
Less Transfer to Endowment	1,634	17	5			
				3,595	3	3
				£60,703	6	6

ASSETS.

	£	s.	d.
The Union Bank of Australia Limited—Balance at Credit	1,936	18	11
The Bank of Australasia—Balance at Credit	35	0	8
Endowment Investments	57,073	2	7
Gordon Craig Library—at cost	1,221	4	4
Library Furniture, Fittings and Equipment—at cost—*less* depreciation	437	0	0
	£60,703	6	6

H. B. DEVINE, President.
BALCOMBE QUICK, Honorary Treasurer.
H. G. WHEELER, Secretary.

Audited and found correct.
YOUNG AND OUTHWAITE,
Chartered Accountants (Australia),
368 Collins Street, Melbourne.
February 12, 1940.

are the vital concern of Fellows and Members of both Colleges who have offered their services in connexion with war work.

Australian Red Cross Society.

On the outbreak of war, the Council decided to offer unoccupied space in the College building to the Commonwealth Government for use during the time of national emergency. This offer was gratefully accepted by the Australian Red Cross Society through the Commonwealth Government. The society now occupies portion of the College building for the purpose of housing its central administrative staff.

Balance Sheet and Accounts.

Copies of the balance sheet and accounts are available for inspection, and Fellows will be asked at this meeting formally to adopt them. Any information concerning the accounts will be supplied on request. The accounts will be published in the next issue of THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY.

Election of Council.

Six nominations were received to fill six vacancies on the Council. All the retiring members of the Council were renominated.

Gifts to the College.

The Council desires to express its gratitude to the following donors:

G. R. A. Syme: A very fine piece of statuary by Benzoni, depicting Andromeda and Perseus.

Dr. A. E. Rowden White: Some of the manuscripts and lantern slides of the late R. Hamilton Russell.

H. G. WHEELER,
Secretary.

COUNCIL MEETING.

THE Council met on Saturday, April 20, 1940, and the following matters arising out of the meeting are published for the information of Fellows.

Election of Council.

Six nominations were received for the six vacancies on the Council. All the retiring members of the Council were renominated and declared reelected. The members of the Council reelected are as follows: E. D. Ahern, George Bell, T. E. Victor Hurley, A. L. Kenny, Sir Henry Newland, Balcombe Quick.

Appointment of Executive Committee.

The following members of the Council were appointed an Executive Committee: Sir Hugh Devine, Sir Alan Newton, A. L. Kenny, T. E. Victor Hurley and Balcombe Quick.

Appointment of State and Dominion Committees.

The following appointments were made:

New South Wales: A. J. Aspinall, V. M. Coppleson, B. T. Edye, J. W. S. Laidley, A. M. McIntosh, I. Douglas Miller, F. Brown Craig.

Queensland: J. C. Hemsley, A. E. Lee, B. T. Mayes, H. S. McLelland, J. J. Power, Neville G. Sutton.

South Australia: R. M. Glynn, I. B. Jose, L. C. E. Lindon, P. S. Messent, B. H. Swift, L. A. Wilson.

Tasmania: F. W. Fay, J. Bruce Hamilton, D. W. L. Parker.

Victoria: W. A. Hailes, A. Fay Maclure, J. Newman Morris, Henry Searby, C. Gordon Shaw, John H. Shaw, B. T. Zwar.

Western Australia: F. J. Clark, H. B. Gill, D. D. Paton.

Dominion of New Zealand: Sir Carrick Robertson, Sir Hugh Acland, David Whyte, D. S. Wylie, P. S. Foster, J. A. Jenkins.

Appointment of State and Dominion Hospital Committees.

The following appointments were made:

New South Wales: A. J. Aspinall, A. S. D. Barton, T. Hamilton, C. B. Howse, J. W. S. Laidley, T. W. Lipscomb, I. Douglas Miller, J. S. MacMahon, Clarence Read, H. H. Schlink, J. C. Storey, Sir Robert Wade, C. E. Winston.

Queensland: E. D. Ahern, J. C. Hemsley, A. E. Lee, H. S. McLelland, Neville G. Sutton.

South Australia: A. M. Cudmore, H. M. Jay, I. B. Jose, Sir Henry Newland, T. G. Wilson.

Victoria: A. E. Brown, Sir Hugh Devine, T. E. Victor Hurley, A. Fay Maclure, J. Newman Morris, Sir Alan Newton, C. Gordon Shaw, Henry Searby, B. T. Zwar.

Western Australia: J. P. Ainslie, F. J. Clark, H. H. Stewart.

Dominion of New Zealand: F. S. Batchelor, F. Gordon Bell, P. Stanley Foster, Sir Carrick Robertson, David Whyte, D. S. Wylie.

Admission of New Fellows.

The following new Fellows were admitted by the Council:

General Surgery.

New South Wales: Wilfrid John Robert Nickson, Kenneth William Starr, John Langston Watt.

Queensland: Malcolm Wesley Carseldine, Kenneth Barron Fraser.

Victoria: John Strahan Bothroyd, Robert Sutherland Lawson, Robert Officer.

New Zealand: Joseph John Brownlee, Ernest Young Comrie, Clifford Samuel James, Herbert Kenneth Pacey, Alan Edwin Park.

Ophthalmology.

Victoria: John Henry Niall.

HYDATID REGISTRY.

THE following is the annual report of the Hydatid Registry of the Royal Australasian College of Surgeons for the year ended December 31, 1939, submitted by Sir Louis Barnett, the Registrar-in-Chief.

The records on file number 1,455, an increase of 151 on the figures reported last year.

The index cards with précis are tabulated in alphabetical order and classified in groups according to the organs mainly responsible for the clinical manifestations. Australian and New Zealand cases are indexed separately.

Records in which the patient's name is not given are indexed in corresponding groups under the letter X.

It must, however, be borne in mind that quite a large proportion of hydatid cysts are multiple in location. For example, in the records of the 857 patients indexed under the heading of liver cysts there are references in 148 of them (17%) to cysts in other regions, mostly peritoneal (98), pulmonary (36), splenic (9), renal (5).

A detailed investigation of the 1,455 records shows that cysts were located in the regions shown in Table II.

Dévé, from his count of collected records, estimated that about one-third of the patients harbour more than one hydatid cyst. Dew thinks the frequency of multiplicity is far higher than that—even double this figure. Racic, of Split, Dalmatia, records 20% multiplicity in 354 cases personally operated on between the years 1914 and 1937.

Mortality.

Anything approaching to accuracy in mortality percentages cannot be gathered from the tabulations, which show 169 deaths out of 1,455 cases (roughly 12%). This is clearly an under-estimate.

The great majority of the records are concerned with hospital patients, and the deaths from hydatid disease are noted only when such deaths occur in the hospital. It is well known that many patients discharged from hospitals die in their own homes from the effects of the hydatid infection, and their deaths are not noted in the hospital records.

A striking example of this inaccuracy is seen in connexion with spinal hydatids. These in the nature of things are invariably fatal sooner or later, yet the registry records 20 cases with only three deaths. My own personal investigations in New Zealand indicate that the mortality rate from hydatid disease is approximately 15%.

TABLE I.

Site.	Total of Records.			Deaths.
Liver	Australia	478		
	New Zealand	379		
			857	113
Multiple abdominal and pelvic nearly all secondary to primary liver cysts	Australia	68		
	New Zealand	48		
			116	12
Lung	Australia	140		
	New Zealand	158		
			298	25
Muscles and fascia	Australia	34		
	New Zealand	39		
			73	2
Bone—				
Spine	Australia	16		
	New Zealand	4		
			20	3
Other bones	Australia	12		
	New Zealand	10		
			22	2
Kidney	Australia	12		
	New Zealand	22		
			34	5
Spleen	Australia	12		
	New Zealand	3		
			15	3
Brain	Australia	4		
	New Zealand	8		
			12	4
Heart	Australia	..	2	
Thyreoid	New Zealand	..	2	—
Parotid	New Zealand	..	1	—
Prostate	New Zealand	..	1	—
Pancreas	New Zealand	..	1	—
Breast	New Zealand	..	1	—
Total			1,455	169

TABLE II.

Location of Hydatid Cysts in 1,455 Cases, in 212 (15%) of which Cysts in more than One Organ or Tissue were Definitely Noted.

Site.	Number in which Cysts were Noted in More than One Organ or Tissue.	Approximate Percentage.
Liver cysts in 899—approximately 62% of all cases. Multiple abdominal and pelvic secondary to liver	225	16
Lung	341	24
Muscles and fascia	77	6
Bone—		
Spine 20		
Other bones 22		
	42	3
Kidney	39	3
Spleen	24	2
Brain	12	1
Heart	2 } rarities	—
Thyreoid	2	
Parotid	1	
Breast	1	
Prostate	1	
Pancreas	1	—

An interesting observation on which Dr. J. B. Cleland, of Adelaide, has laid stress is that in the records of autopsies something like 2% of the patients are shown to have harboured hydatid cysts, at least half of which were unsuspected during life.

Grateful Appreciation of Help.

Again I thank Mr. H. G. Wheeler (Secretary of the College), the local registrars in the various States and in the Dominion, medical superintendents, medical practitioners, house surgeons and others, who have kindly helped me to secure records during the last year. In this connexion, I wish to mention two names particularly: one is Dr. J. B. Cleland, of Adelaide, who placed at my disposal a selection of his large and valuable collection of clinical and post-mortem records, and the other is Dr. J. Bruce Hamilton, of Hobart, whose keen efforts have led to the acquisition of a considerable number of new Tasmanian cases.

L. E. BARNETT,
Hydatid Registrar-in-Chief.

Notices.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to pages xx and xxx among the advertisements, which illustrate some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. The publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

Editorial Notices.

EDITORIAL communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

Reprints can be supplied at cost price; the minimum number is fifty copies. Orders for reprints must be given when the proof is returned.

Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, Victoria, Australia.

Business communications and remittances should be addressed to Butterworth and Co. (Australia) Ltd., 6-8 O'Connell Street, Sydney.